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BULLOUS KERATITIS

WITH PARTICULAR REFERENCE TO THE PATHOLOGY OF EXPERIMENTAL CORNEAL VESICULATION

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BOSTON

. . . wir müssen die anatomischen und die functionellen Veränderungen, welche sich während des Lebens feststellen lassen, mit den Leichenbefunden in den successiven Entwicklungsphasen vergleichen und, so weit das möglich, auch Experimente an Thieren zu Hilfe nehmen, um irriger Deutung des Befundes im Leben und im Tode möglichst vorzubeugen.*

From C. F. von Arlt's *Zur Lehre vom Glaucom,*
Vienna, W. Braumüller, 1884, p. 1.

The experimental production of a condition similar to that known clinically as bullous keratitis was first reported about one year ago.[1] This was followed by a report of certain practical observations pertaining to corneal edema.[2] The assumption was made that both the experimental and the clinical condition have the same basic pathogenesis, consisting of osmotic attraction of water into the corneal epithelium from the tears or from other fluid outside the eye. The similarity of the two conditions is evidenced not only by their gross appearances but by the fact that they respond in like manner to changes in the osmotic tension of the fluid outside the eye. It is my present purpose to describe the pathologic picture of the experimental condition and to compare it with that of clinical bullous keratitis.

The pathogenesis of experimental bullous keratitis is important so far as a similar process occurs in cases of clinical bullous keratitis.

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* "We must compare the anatomic and functional changes which can be determined during life with the autopsy observations in the successive developmental phases, and also, as far as is possible, we must get help from animal experimentation in order to avoid erroneous interpretation of the findings in life and in death."

1. Cogan, D. G.: Experimental Production of So-Called Bullous Keratitis, Arch. Ophth. **23**:918 (May) 1940.

2. Cogan, D. G.: Some Practical Observations Pertaining to Corneal Edema, Arch. Ophth. **25**:552 (April) 1941.

Yet, with such material as is available, it would be surprising if the pathologic pictures were identical in the two conditions, for the experimental process is necessarily acute, while the clinical process is customarily chronic. Such material as is obtainable for study of the latter condition reveals chiefly late results. In a case to be described subsequently in this report—the earliest histologic study of bullous keratitis on record—the condition was not older than three weeks, but even in this case secondary changes had occurred to mask the initial process. The eyes with experimental bullous keratitis, on the other hand, were removed in less than an hour after the onset of the condition. There is nevertheless a striking similarity in the basic changes in histologic sections in the two conditions, and for the purpose of comparison they will be discussed together. It will be evident, among other things, that certain pathologic structures which have given rise to speculation and difference of opinion among pathologists may now be given a rational explanation in view of the early changes.

HISTORICAL SURVEY

Past developments which are responsible for current ideas regarding the pathogenesis of bullous keratitis are not many. Von Graefe [3] was the first to report a histologic study of bullous keratitis, but his study was based on the examination of the anterior wall of only one bulla, and in reporting that this wall was made up of epithelium, Bowman's membrane and corneal stroma he differed from all subsequent investigators. It is now agreed that the anterior wall is made up of epithelium alone or of epithelium and cells resembling those of connective tissue.

Leber [4] was the first to report the histologic study of a whole eye with bullous keratitis. The following year Fuchs made a similar study.[5] Leber's priority is commonly overlooked (indeed, Fuchs claimed that he made the first study of bullous keratitis on the whole eye), owing, I presume, to the fact that Leber's notes on the pathologic picture were incidental to a larger study, dealing with intercellular spaces in the epithelium. However, in Leber's description of the epithelial changes in glaucomatous and staphylomatous eyes there can be little doubt that

3. von Graefe, A.: Notiz über Bläschenbildung auf der Hornhaut, Arch. f. Ophth. (pt. 1) **2**:206, 1855.

4. Leber, T.: Ueber die intercellularen Lücken des vorderen Hornhaut-Epithels im normalen und pathologischen Zustande, Arch. f. Ophth. (pt. 1) **24**:252, 1878.

5. Fuchs, E.: Keratitis bullosa, Ber. ü. d. Versamml. d. ophth. Gesellsch. **12**:212, 1879.

it was bullous keratitis which he was discussing. He spoke of vesicular degeneration of the epithelium, and he reported that in many of the sections observed by him the deepest cells were loose or absent, while the rest of the epithelium was separated from Bowman's membrane.

From the time of Leber and Fuchs to the present, numerous individual cases have been reported and suggestions made as to the pathogenesis of bullous keratitis. Rather than discuss these in chronologic order, I shall mention the main theories arrived at by a perusal of the literature and present only such facts as are necessary for the evaluation of the theories.

The theories, with minor variations, are fourfold: first, that the epithelial changes are due to percolation of fluid from the aqueous through the cornea into the superficial layers; second, that the epithelial changes are the histologic expression of a neurodystrophy; third, that the epithelial changes are secondary to disease of Bowman's membrane, and fourth, that, as I have proposed, the epithelial changes are due to an osmotic imbibition of fluid from the tears.

The theory that the epithelial changes result from percolation of fluid into the epithelium through its posterior surface is the one most widely held. It was first suggested by Saemisch [6] and was later elaborated by Leber [4] and by Fuchs.[7] Saemisch expressed the belief that the cause for the forward percolation of fluid is obstructed drainage of lymph in the corneal periphery. Leber concluded that fluid is pressed mechanically out of the cornea into the epithelium. Fuchs maintained that the source of the fluid is the aqueous and that its presence in the stroma is only in transit. It is rather paradoxic that the chief evidence cited by Fuchs for his contention was Leber's experimental data on the removal of endothelium. Leber himself did not draw any analogy between his experimental condition and bullous keratitis, and he criticized Fuchs for inferring that the infusion of dyes was indicative of fluid transfer.[8] Nevertheless, Fuchs's description was the first thorough one of the objective pathologic changes associated with bullous keratitis, and the majority of subsequent investigators, in confirming his pathologic observations, have also accepted the pathogenesis which he suggested. Authors who have expressed agreement with this general theory of the forward percolation of fluid into the epithelium are Landesberg,[9]

6. Saemisch, E. T.: Krankheiten der Conjunctiva, Cornea und Sklera, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1876, vol. 4, pt. 2, chap. 3, p. 271.

7. Fuchs, E.: Ueber glaukomatöse Hornhauttrübung, Ber. ü. d. Versamml. d. ophth. Gesellsch. **13**:73, 1881.

8. Leber, T., in discussion on Fuchs.[7]

9. Landesberg, M.: On Keratitis Bullosa, Arch. Ophth. & Otol. **6**:135, 1877.

von Arlt,[10] Michel,[11] Bock,[12] Brügger,[13] Meller,[14] Wirths,[15] Gilbert [16] and Rones.[17]

Brügger and later Gilbert pointed out that the bullae are not composed of fluid alone but contain broken-down cellular material, an observation to which I shall return. The breakdown of cells, according to Brügger is due to the stasis of a fluid, which he believes to be nutritive corneal fluid. Presumably this results in an artificial stimulation of the epithelium with the formation of cells having an abnormally low vitality. Gilbert expressed the belief that the disintegration results from inadequate nutrition.

The second theory, explaining the epithelial changes on the basis of a neurodystrophy, is concerned less with the altered water metabolism, which admittedly occurs in the corneal epithelium, than with the basis for this disturbance. This theory, therefore, may overlap the first. Although Schnabel [18] had previously hypothecated that glaucoma results from a neurotic hypersecretion, the first to maintain that the changes associated with bullous keratitis represent a neurotrophic disturbance were Birnbacher and Czermak.[19] They claimed that the same type of change occurs in the skin with herpes zoster and with pemphigus. Like the cutaneous changes, the changes associated with bullous keratitis are, according to these authors, due to neuritis. More recently, Elschnig [20]

10. von Arlt, C. F.: Klinische Darstellung der Krankheiten des Auges, Vienna, W. Braumüller, 1881, p. 125.

11. Michel, J.: Lehrbuch der Augenheilkunde, Wiesbaden, J. F. Bergmann, 1884, p. 264.

12. Bock, E.: Beitrag zur pathologischen Anatomie der Cornea, Klin. Monatsbl. f. Augenh. **24**:443, 1886.

13. Brügger, R.: Ein anatomischer Beitrag zur Lehre von der Keratitis bullosa, Klin. Monatsbl. f. Augenh. **24**:500, 1886.

14. Meller, J.: Die Sclerose der Kornea, Klin. Monatsbl. f. Augenh. **43** (pt. 2):209, 1905.

15. Wirths, M.: Beiträge zur Anatomie der Keratitis vesiculosa und bullosa, Ztschr. f. Augenh. **16**:99, 1906.

16. Gilbert, W.: Klinische und pathologisch-anatomische Beiträge zur Kenntnis degenerativer Hornhauterkrankungen: I. Pannus degenerativus und Keratitis bullosa, Arch. f. Ophth. **69**:1, 1909.

17. Rones, B.: Pathologic Conditions of the Cornea: I. Disturbances of the Epithelium, Endothelium, and Bowman's and Descemet's Membrane, Arch. Ophth. **24**:374 (Aug.) 1940.

18. Schnabel, J.: Beiträge zur Lehre vom Glaukom: I, Arch. f. Augen- u. Ohrenh. **6**:118, 1878.

19. Birnbacher, A., and Czermak, W.: Beiträge zur pathologischen Anatomie und Pathogenese des Glaukoms, Arch. f. Ophth. (pt. 2) **32**:1, 1886.

20. Elschnig, A., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologsichen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, p. 881.

stated that bullous keratitis is due, in the case of glaucoma at least, to a dystrophy resulting from glaucomatous changes in the corneal nerves. As evidence for the neurogenic origin, Elschnig pointed to the hypesthesia that is a constant accompaniment of the condition. Of particular interest from my point of view is the suggestion made by Elschnig that the trophic disturbances result in a chemical change in the cells and that these cells then take up fluid not only from adjacent cells but possibly from tear fluid as well. Salzmann [21] also stated that bullous keratitis is analogous to herpes of the cornea. This fact, together with the island-like distribution which he alleged occurs about the nerves, led him to the belief that the essential disturbance in cases of bullous keratitis with glaucoma is a dystrophy. No explanation was given by any of these authors to account for the absence of bullous keratitis with trigeminal lesions.

The theory that the primary disturbance in cases of bullous keratitis is alteration of Bowman's membrane has for some time been associated with the name of Ewing.[22] This author observed in histologic examination of 1 eye fatty droplets in Bowman's membrane. These, he stated, antedated the epithelial changes; he therefore concluded that such an observation presumably represents the initial change in all cases. More recently, Allen,[23] after studying a number of specimens histologically, proposed that the essential pathologic process of bullous keratitis is a change in Bowman's membrane that interferes with its giving a foothold to the epithelial cells. The evidence that Allen presented was the fact that the whole epithelium in the sections is commonly found loosened from Bowman's membrane. Difficult to reconcile with the theory that Bowman's membrane is the seat of the disease is the report of a large number of cases in which Bowman's membrane was found to be normal.

Finally, there is the theory that corneal vesiculation and bullous keratitis are dependent on osmotic hypertonicity of the cornea relative to the fluid bathing the eye. According to this theory, fluid is then imbibed into the epithelium from its anterior surface backward instead of in the reverse direction as is commonly believed. This imbibition of fluid from in front certainly is the mechanism of experimental bullous keratitis, and what evidence is available points to a similar mechanism for clinical bullous keratitis.

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21. Salzmann, M.: Die glaukomatöse Hornhautentartung, Arch. f. Ophth. **139**:413, 1938.

22. Ewing, A. E.: Bullous Keratitis: Fatty Degeneration of Bowman's Membrane, Am. J. Ophth. **21**:161, 1904.

23. Allen, T. D.: On the Pathology of Bullous Keratitis, Tr. Am. Ophth. Soc. **30**:391, 1932.

PATHOLOGIC PICTURE

The pathologic picture of experimental bullous keratitis is presented in the following sections, together with correlated observations pertaining to the clinical condition.

Preparation of Material.—Except when otherwise noted, all the eyes used in this study were cats' eyes, and with a single exception the corneal vesiculation had been produced in vivo. As was described in a previous communication, the method of producing the corneal vesiculation consisted in injecting salt solutions into the anterior chamber. Concentrations of 2.5, 5, 10 and 20 per cent were used. The surface of the cornea, which was contaminated as little as possible by the salt solutions, was kept moist either with the tears or with the tears and physiologic solution of sodium chloride.

Gross evidence of corneal changes began to appear within five minutes after the injection. The stronger the concentration of the salt solution injected, the more quickly did the changes occur and the more profound was the vesiculation. The first change appeared to be edema of the epithelium and superficial opacity. The superficial position of the opacity was evident even without slit lamp examination, for slight scratches on the surface of the cornea left corresponding clear lines. After an interval varying from five to fifteen minutes, depending again on the concentration of the salt solution, vesicles appeared in the region where the edema had been most intense. These were at first discrete but increased in size as one watched them. As the process continued the vesicles became confluent to form large bullae and ultimately separated the entire epithelium. With the weaker concentrations the corneal changes never passed beyond the stage of edema or beyond the production of a solitary vesicle. The most profound changes were produced, on the other hand, by injecting 20 per cent salt solution into the anterior chamber of a freshly enucleated eye and then immersing the whole globe in distilled water.

An abnormal looseness of the epithelium was noticed in all the eyes on sectioning, even though in all instances it was not grossly apparent in the intact eye.

Contrary to my previous impression obtained from experiments on dead eyes, preliminary removal of the endothelium altered the course of experimental bullous keratitis. When the endothelium of the living cornea had been scratched prior to injection of the salt solution, the epithelium overlying the scratched portion showed edema and vesiculation first. This was not the case in eyes taken from animals which had been dead for several hours but was true of eyes freshly enucleated from living animals. I surmise that in the eyes of dead animals the endo-

thelium is such an ineffective barrier that its local removal does not produce a localized effect.

All the eyes, with 1 exception, were fixed in solution of formaldehyde and embedded in celloidin (a pyroxylin preparation). The whole eye was fixed intact in the earlier experiments, but in the later experiments it was found more satisfactory to bisect the globe at the equator and to fix only the anterior half after removal of the lens. The usual eosin-hematoxylin stains were used for most of the sections, although a few were stained by the modified Gram method to study the subepithelial debris.

Histologic Observations.—In the presence of experimental corneal vesiculation the epithelium as a whole is increased in thickness, in places to two or three times the normal. This is due to swelling of the individual cells and not to increase in their number, for the epithelium retains the normal number of layers. The swelling involves not only the cytoplasm but the nuclei. Instead of remaining spindle shaped, the nuclei become ellipsoid or circular. With the swelling there is a decrease in the staining intensity of both the cytoplasm and the nuclei.

Although this change in volume may affect all the epithelial layers uniformly, it frequently leaves strata of relatively less swollen cells. These stand out by their more darkly staining cytoplasm and by their small, dark and elongated nuclei (fig. 1*A*). In other words, the strata of less swollen cells look approximately like the normal epithelium. They usually occupy one to two cell layers and course parallel to the surface of the epithelium, although occasionally they may course obliquely across its thickness.

Stratification similar to that found in the experimental condition occurs in human eyes with bullous keratitis (fig. 1*B*). Fuchs [25] noted that edema may select certain layers of the epithelium and may produce an apparent alternation of swollen and relatively unswollen layers. Elschnig [20] observed that, while some layers were swollen, the others were actually shrunken, and he suggested that the fluid had been transferred from the one to the other. Salzmann,[21] agreeing with Elschnig, expressed the opinion that the shrinking was the more striking abnormality. As has been mentioned, however, the strata of less swollen cells in experimental bullous keratitis are the ones which are more nearly normal and appear shrunken only by contrast with the strata of swollen cells.

In many sections of corneas with experimental bullous keratitis and occasionally in those with clinical bullous keratitis the superficial layer is the most faintly stained of all, and not infrequently its cytoplasm is separated from that of the underlying cells by an abrupt transition

24. Footnote deleted.

25. Fuchs, E.: On Keratitis, Tr. Ophth. Soc. U. Kingdom **22**:15, 1902.

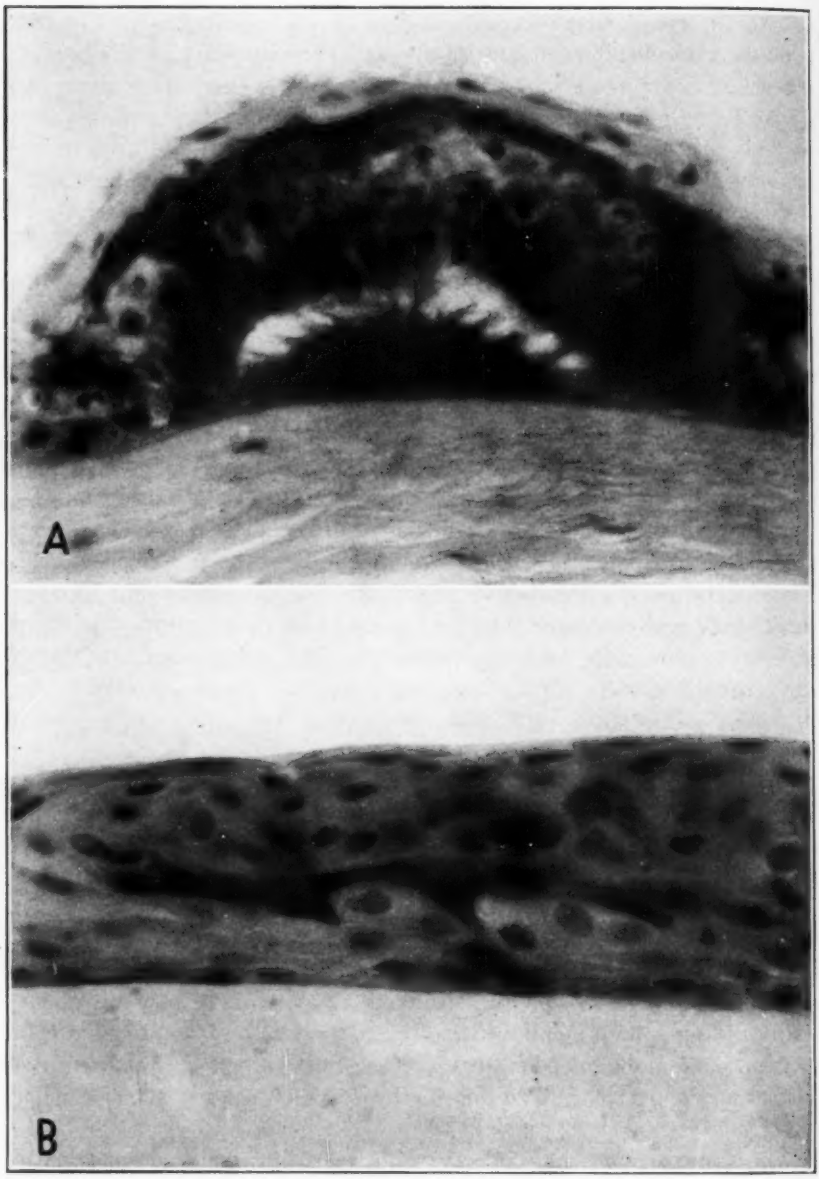

Fig. 1.—*A*, typical bulla of experimental bullous keratitis. Noteworthy are the strata of relatively less swollen cells and the debris beneath the epithelium. High power magnification. *B*, epithelium from an eye with clinical bullous keratitis, showing strata analogous to those in *A*. High power magnification.

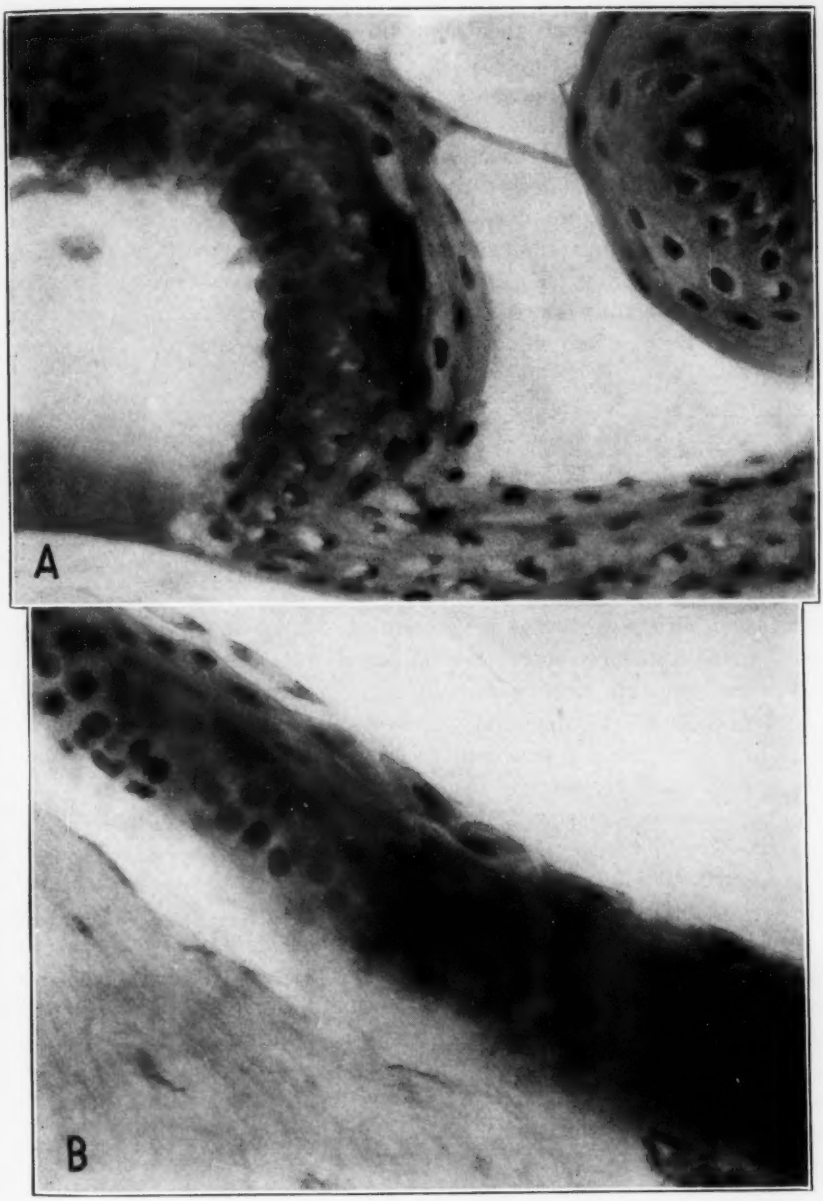

Fig. 2.—*A*, portion of the epithelium in a case of experimental keratitis, showing preferential swelling of the superficial cells and vacuolation of some nuclei. High power magnification. *B*, swelling of the superficial cells in a case of bullous keratitis. High power magnification.

(figs. 1*A* and 2). The nuclei of this superficial layer are distinctly larger than those of the other layers, and the cytoplasm is obviously swollen. Although such an abrupt difference between the surface and the other layers is never seen in the normal cat's eye, it apparently represents a process analogous to what I have observed takes place normally. If one examines sections of a normal cat's eye with high power, here and there on the surface appear ghostlike cells, the cytoplasm of which stains very faintly if at all and the pale nuclei of which are swollen to several times that of the underlying cells (fig. 3*A*). These ghost cells are elevated above the outer convexity of the epithelium, but in places their posterior surfaces form small, shallow craters in the underlying layers, their point of contact with the rest of the epithelium. I know of no explanation for this change in the superficial cells other than that they imbibe fluid prior to being dislodged. Whereas in the normal eye this process is seen to affect at any one time only a few isolated cells on the surface, in the presence of experimental bullous keratitis it may affect one or two whole layers. Conspicuous ghost cells may also be produced by instilling tap water on the normal cornea (fig. 3*B*).

The middle epithelial layers in the presence of experimental bullous keratitis show most commonly a uniform swelling of the nuclei and the cytoplasm, although, as has been mentioned, they may show stratification. Less commonly there may be islands of swollen cells, occupying not more than four or five cells in any one area.

A nuclear change that may occur in all the layers but occurs perhaps with greatest frequency in the middle epithelium is vacuolation. Here the nuclear material, instead of being uniformly enlarged, is crowded into one portion of the nucleus by the presence of one or more vacuoles (fig. 4*A*). In most instances the chromatin material is pushed to one pole of the nucleus so that it rests like a cap on the vacuole, but the clear vacuole may be in the center of the nucleus with a thin ring of nuclear material in the periphery, or two vacuoles may be present, compressing the attenuated nuclear material between them. Various other bizarre shapes may be assumed by the nuclear material. Although these vacuoles may appear to be in the cytoplasm, it seems to me more likely that they are in the nuclei, because of their apparent intimacy with the nuclear fragments. Also, their sharp demarcation from the cytoplasm suggests that they are separated by a membrane, such as the nuclear wall.

These vacuoles in the nuclei of eyes with bullous keratitis were described in detail by Leber,[4] and his observations were confirmed by de Schweinitz and Shumway.[26] Leber stated that they were similar

26. de Schweinitz, G. E., and Shumway, E. A.: Ueber die Histologie der Keratitis bullosa bei glaucomatösen Augen, Arch. f. Augenh. **52**:349, 1905.

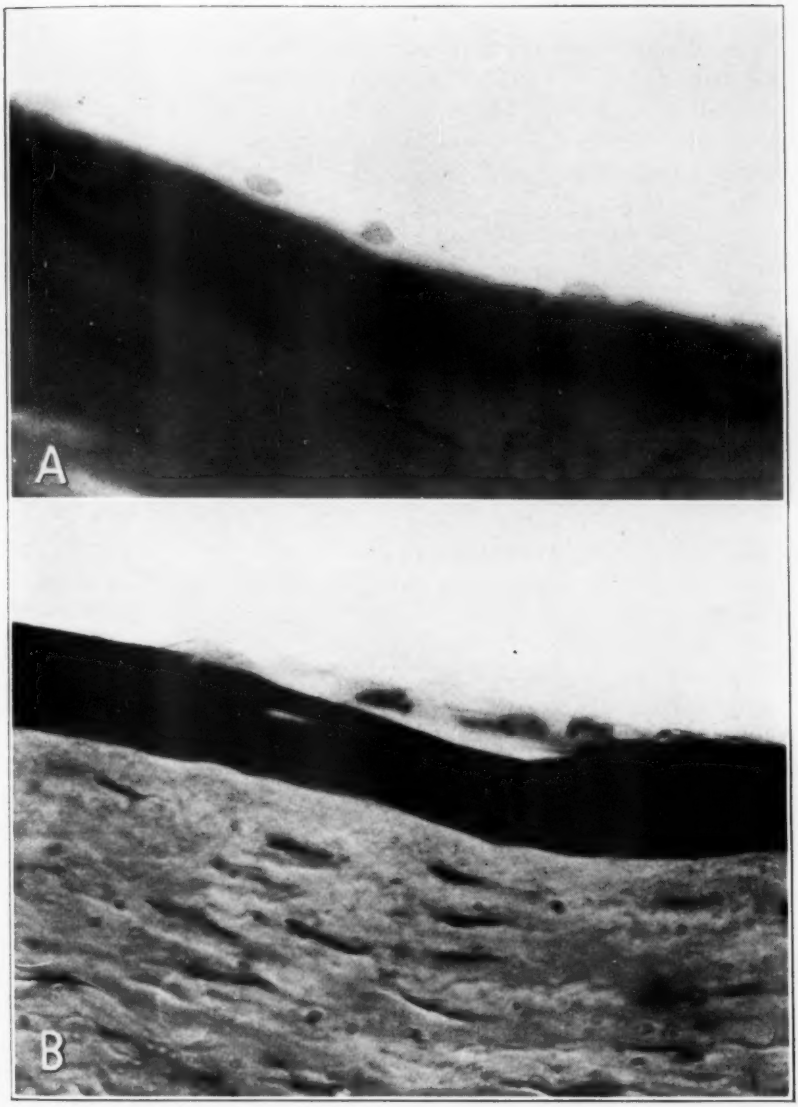

Fig. 3.—*A*, overexposed picture of normal cat's epithelium, showing nuclei of ghostlike cells on the surface. High power magnification. *B*, exaggeration of ghost cells produced by keeping the cornea moist with tap water. High power magnification.

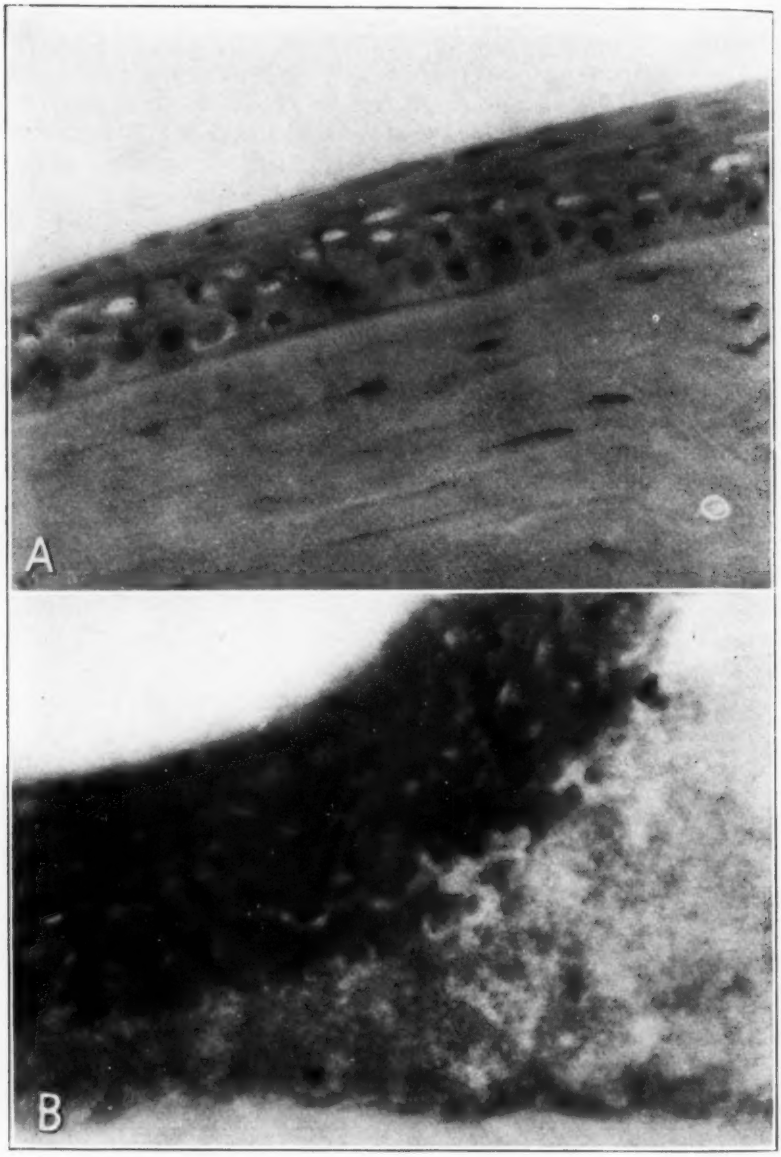

Fig. 4.—*A*, epithelium in a case of experimental bullous keratitis, showing vesiculation of the nuclei. High power magnification. *B*, epithelium and subepithelial debris in a case of experimental bullous keratitis, showing disintegration of the basal cells. A single free nucleus is visible in the mass. High power magnification.

to nuclear changes which he had produced by injecting oil of turpentine into the cornea, but most noteworthy is his observation that similar changes in the nuclei can be produced by previous immersion of the cornea in distilled water or tap water.

Most significant are the changes that occur in the basal cells. Here the cells are swollen, with or without vacuolation of the nuclei. The cell walls stand out distinctly in some sections. At certain places, however, where the epithelial swelling is especially marked the walls of the basal cells are not visible; the nuclei and the cytoplasm merge into an amorphous mass containing not only vacuoles but debris, which stains like cytoplasm (fig. 4*B*). Commonly there are large clear areas that appear empty in the sections. The epithelium may be elevated in some places only slightly and in other places so much that there can be no doubt of the section's passing through a bulla. On the whole, small bullae contain the debris, while the large bullae contain clear areas in which the debris is crowded to one corner. In the middle-sized bullae the debris may be scattered throughout the space betwen the epithelium and Bowman's membrane, but it is not so dense that it alone could account for the separation of the epithelium. The large bullae give the impression that the clear areas had been occupied by fluid that was lost in fixing or in sectioning.

At first sight this subepithelial material appears to be exudate or transudate. It would be surprising for either of these substances to be so conspicuous in this portion of the cornea within the short period of ten to fifteen minutes. It would be well nigh inexplicable for no trace of the exudate or transudate to be found in the corneal stroma through which it must have passed to reach the subepithelial region. Furthermore, the morphologic and staining characteristics of this debris differ from those of fibrin. Subepithelial debris is granular and stains like the cytoplasm of the epithelial cells, while exudate or transudate, seen, for instance, in the anterior chamber of the same eye, is fibrinous, staining only faintly and consisting of thousands of tiny threads with a few enmeshed red and white cells (fig. 5). The subepithelial debris does not stain with the modified Gram stain as does the material in the anterior chamber. Finally, that it is not hematogenic material would appear to be proved by the fact that the subepithelial substance is present when bullous keratitis is produced in enucleated eyes.

There is a more obvious and, to my way of thinking, more tenable explanation for the subepithelial debris, based on the interpretation of the substance as disintegrating epithelial cells. Apparently the swollen basal cells ultimately burst and disgorge their contents into the space beneath the epithelium, loosening and separating it for a variable distance from the superficial stroma. In some sections the actual transition of cellular cytoplasm into the amorphous mass can be seen (fig. 4*B*).

The nuclei in various stages of fragmentation may be found free in this mass (fig. 6*A*). They may be irregularly shaped when they come from an area of nuclear vacuolation, or they may be free, floating, round bodies resembling lymphocytes. None of this amorphous mass is to be found in the stroma. Analogous sections taken in cases of clinical bullous keratitis can be found which are amenable to the same explanation (fig. 6*B*). In tracing the origin of this subepithelial debris in the early stages of experimental bullous keratitis, I believe, there lies an explanation for a membrane that has aroused the speculation of clinical pathologists. Leber [4] described a newly formed membrane between the

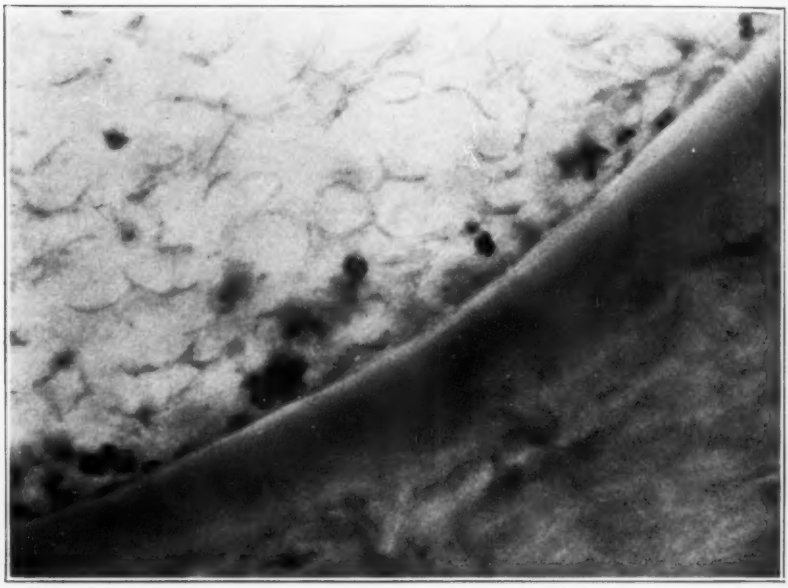

Fig. 5.—Posterior portion of the cornea in a case of experimental bullous keratitis, showing fibrinous exudate in the anterior chamber. This does not resemble the subepithelial debris in figure 4 *B*. High power magnification.

epithelium and Bowman's membrane in glaucomatous eyes with bullous keratitis. He said that it is unlike pannus in that vessels are often absent and the membrane has been at one time fluid or very soft, for it has molded itself to the epithelial cells. Leber expressed the opinion that wandering corneal corpuscles come into the subepithelial tissue through Bowman's membrane and form connective tissue. Fuchs,[27] describing the subepithelial substance in more detail, said that it is in the beginning uniformly granular and probably comes from the aqueous,

27. Fuchs (footnotes 5 and 7).

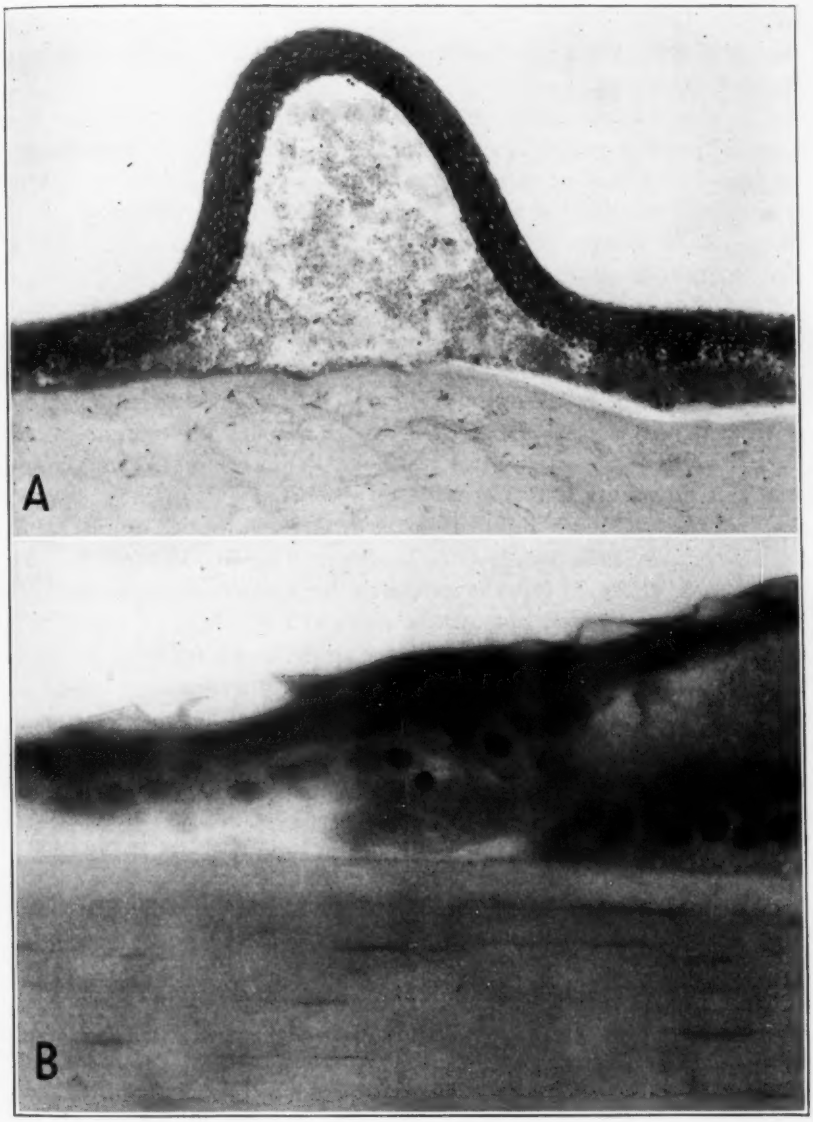

Fig. 6.—*A*, bulla of experimental bullous keratitis, showing granular debris and free-floating nuclei beneath the epithelium. Low power magnification. *B*, subepithelial debris and one free-floating nucleus in a clinical case of bullous keratitis. High power magnification.

which percolates through the cornea and spreads out under the epithelium. Here it forms what he called the "accessory membrane." It differs from physiologically structureless membranes by its extraordinary fragility. Gradually it is invaded by cells, which in his opinion come from the periphery. With the formation of connective tissue the membrane partly disappears.

Fuchs's objective observations in regard to the membrane have been repeatedly confirmed. That broken-down epithelial cells contribute to the substance was suggested by Brügger [13] and Gilbert.[16] Brügger expressed the belief that the cells break down because the newly formed tissue has an inherently lower vitality, while Gilbert stated the opinion that the cells break down because of unfavorable mechanical conditions and inadequate nutrition.

Following the leadership of Leber and Fuchs, most investigators of clinical bullous keratitis have attributed the formation of connective tissue beneath the epithelium to cells which have come from the stroma or from the periphery. Insufficient consideration would appear to have been given to the possibility that it is epithelial in origin. Allen [23] considered this possibility but rejected it on the ground that "the suggestion that connective tissue cells originate from well-formed adult epithelium is contrary to known laws." However, this would appear not to be the case, as Bakker [28] has shown clearly for the lens epithelium and as Verhoeff [29] has pointed out for the pigment epithelium. I know of no way to prove that this tissue beneath the epithelium is epithelial in origin, but it is a possibility. In any case, the cellular membrane develops late and does not occur in eyes with acute experimental bullous keratitis.

The findings in cases of experimental bullous keratitis do not support the thesis that Bowman's membrane is primarily responsible for the changes. The cat has no Bowman's membrane like that found in the human eye. Indeed, the epithelium appears to be directly juxtaposed to the anterior stroma. Although reference to Bowman's membrane has been frequently made in the various histologic studies of clinical bullous keratitis, especially in confirmation or denial of Fuchs's statement that the canals are dilated, only two persons have proposed that disturbances of Bowman's membrane are primarily responsible for the disease. One was Ewing,[22] who found fatty degenerative changes in the membrane, and the other was Allen,[23] who was impressed by the frequency with which the epithelium is separated from Bowman's membrane. There is no incontrovertible reason to think that these changes

28. Bakker, A.: Eine Methode, die Linsen erwachsener Kaninchen ausserhalb des Körpers am Leben zu erhalten, Arch. f. Ophth. **135**:581, 1936.

29. Verhoeff, F. H., in discussion on Rones, B.: Formation of Drusen in the Lamina Vitrea, Tr. Sect. Ophth., A. M. A., 1937, p. 178.

are primary. The production of bullous keratitis in eyes that have no Bowman's membrane does not substantiate the assertion of the essential role of this membrane in the pathogenesis of the disease.

The stroma in eyes with experimental bullous keratitis shows no marked deviation from the normal. The thickness is increased to some extent, but rarely is it doubled. The stromal nuclei are indistinguishable from the normal. The lamellas are probably increased in thickness, although this is difficult to conclude from the sections. The interlamellar spaces are somewhat distended in cases of marked involvement. These spaces are, however, clear. There is nothing to suggest transudate or exudate in them, nor is there any increase in the leukocyte content of the corneal stroma. If it were maintained that the subepithelial nuclei were lymphocytes instead of disintegrated epithelial nuclei, similar cells might be expected in the stroma.

The fact that there are no marked changes in the stroma of eyes with bullous keratitis has been repeatedly observed, according to histologic reports of clinical cases. This is not difficult to explain by the hypothesis of Leber, who postulated that the stroma contributes passively by having its fluid pressed out and into the epithelium. More difficult to explain by this assumption is the increase in corneal thickness. The absence of appreciable changes in the stroma, however, would not accord with Fuchs's explanation that transudate or exudate, having come from the aqueous, percolates through the stroma into the epithelium. Fuchs stated that, although there is some increase in the contents of the stroma, especially in its anterior layers, there is no substance in the cornea which gives the staining characteristics of the subepithelial debris. This he attempted to reconcile with his theory by assuming that the transudate which comes through the cornea undergoes some fermentative change when it comes in contact with the epithelium. The resultant product, according to him, must have morphologic and histologic characteristics different from the mother substance, which comes through the stroma.

Although in the presence of either experimental or clinical bullous keratitis the stroma is generally little changed, I regularly found a definite swelling of the stroma in those eyes in which I had purposely scratched off part of the endothelium and Descemet's membrane prior to injection of the salt solution. The cornea in the region corresponding to the lesion of the posterior surface became abruptly double or treble its thickness elsewhere (fig. 7). This increase was due, so far as I could tell, exclusively to the swelling of the stroma. Here again the lamellas were probably increased in size, for they stained more lightly and the interlamellar spaces stood out more clearly. The

epithelium over these portions of the cornea did not show the changes of bullous keratitis more than elsewhere, although in the intact eye these areas over the scratched portions were the first to show gross changes. This experiment of scratching the posterior surface of the cornea and of producing acute swelling in the stroma is analogous to the familiar experiments of Leber,[30] except that Leber did not subsequently inject salt into the anterior chamber. Like his experiments, mine show that acute changes occur in the stroma and not in the epithelium.

Descemet's membrane was normal in all eyes with experimental bullous keratitis.

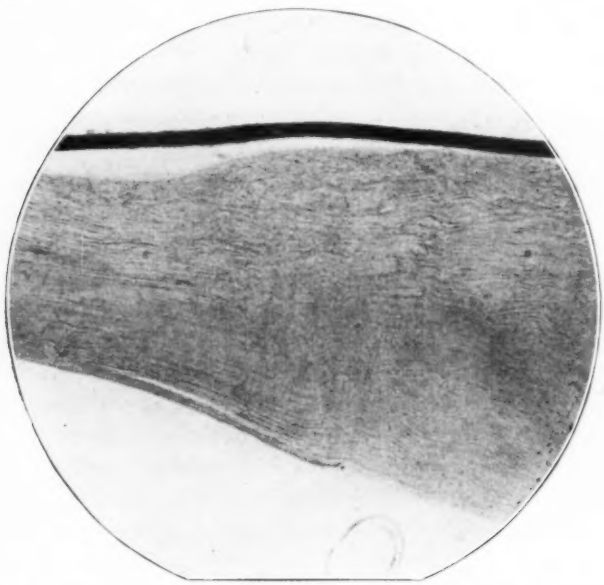

Fig. 7.—Section of the cornea in a case of experimental bullous keratitis, showing abrupt swelling of the stroma in the portion corresponding to a scratch on the posterior surface. Low power magnification.

The endothelium showed no conspicuous deviation from the normal. The nuclei were distinct. The cytoplasm may have been slightly shrunken in those eyes in which salt had been injected into the anterior chamber, but the changes were slight.

The histologic changes of experimental bullous keratitis in cats and of clinical bullous keratitis in human beings are thus basically similar.

30. Leber, T.: Cirkulations- und Ernährungs-Verhältnisse des Auges, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1903, vol. 2, pt. 2, chap. 11, p. 275.

31. Footnote deleted.

As has been said, both are characterized by edema of the epithelium, often involving strata differently. Individual cells are swollen, and the basal cells commonly burst and disgorge their cytoplasm and nuclei beneath the epithelium. In consequence of this and of the collection of a clear fluid from the tears, the epithelium is loosened from its underlying attachment. Like that of so many other biologic processes, the effect is more intense in some places than in others. Where it is more marked the epithelium is elevated to form vesicles and bullae. The experimental and clinical conditions are also similar in their characteristic lack of involvement of the corneal stroma.

There are certain differences between the histologic pictures of the acute experimental and the chronic clinical condition. In the first place, the vesicles in the experimental condition are formed exclusively between the epithelium and the superficial stroma, whereas in the clinical condition some of the vesicles are intraepithelial (fig. 8*A*). Second, in the bullous keratitis of human beings, but not in experimental conditions, a characteristic concentric arrangement of epithelial cells is occasionally found, similar to pearl bodies in epitheliomas. Third, the subepithelial material in the experimental condition is a mixture of amorphous debris and free nuclei, while in the clinical condition there is also an organized tissue containing what appear to be connective tissue cells and also a few blood vessels (fig. 8*B*). None of these differences—so far as I know, they are the only major ones—invalidates the analogy between the experimental and the clinical condition.

The intraepithelial vesicles may arise in one of two ways; either they develop in situ or, having formed between the epithelium and underlying tissue, they are surrounded and pushed forward by the growth of basal cells on the posterior surface. Histologic evidence for either origin may be found. The islands of swollen cells previously mentioned in connection with experimental conditions may be interpreted as the start of an intraepithelial process. On the other hand, the approximation of the basal cells toward each other seen in sections from human eyes may be interpreted as the beginning of a segregation of the vesicles (fig. 9). Whichever way the intraepithelial vesicles arise, the process is undoubtedly the same as that described as occurring in the subepithelial region, for the intraepithelial vesicles contain the same type of protoplasmic debris. I am inclined to believe that the vesicles are all subepithelial in the early stages, for this is the only type seen in examples of experimental vesiculation.

The organization of the subepithelial debris into a cellular membrane would take time and could not be expected to occur in the acute experiments. The debris, like that found in the experimental condition, nevertheless exists in clinical cases in spite of the formation of this

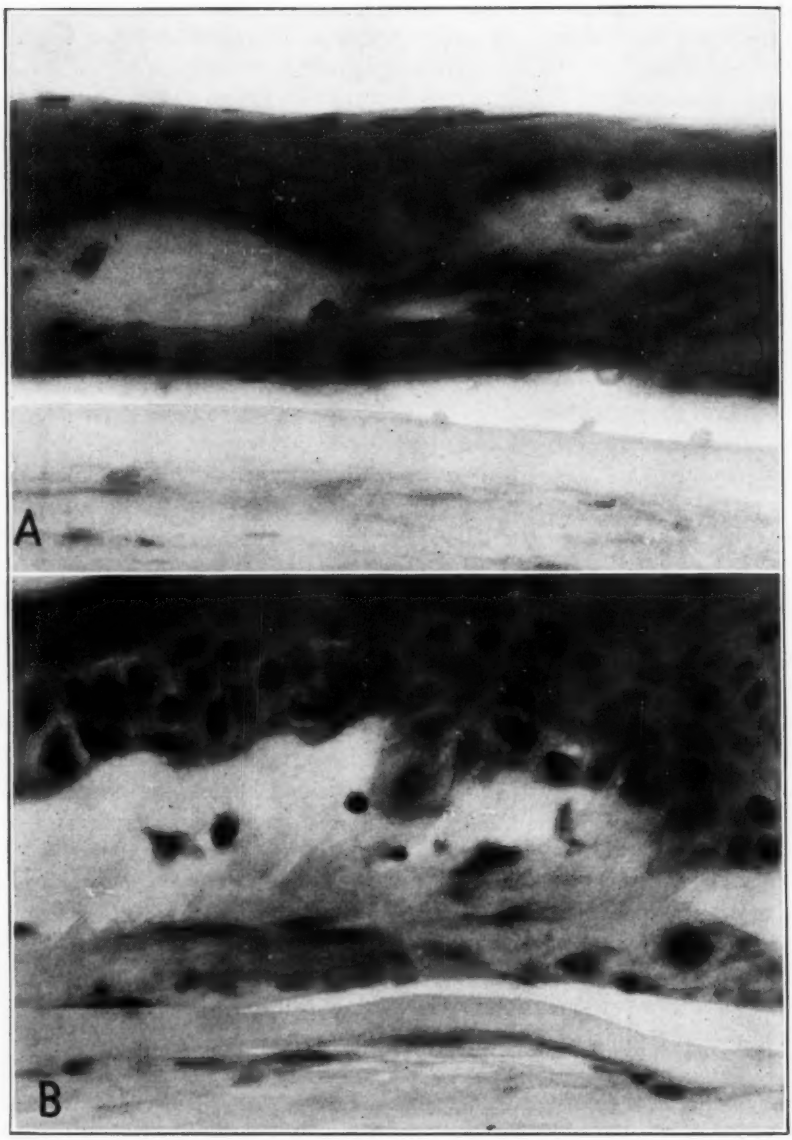

Fig. 8.—*A*, epithelium in a case of clinical bullous keratitis, showing intraepithelial vesicles. High power magnification. *B*, epithelium and subepithelial tissue in a clinical case of bullous keratitis. Note the beginning formation of connective tissue anterior to Bowman's membrane. High power magnification.

membrane. This probably means that the breakdown of the cells continues after organization of the membrane and that persistence of these processes accounts for the frequent deposition of so many layers of connective tissue.

In view of the different durations of the experimental and clinical conditions, it is surprising that there is not a greater difference in the histologic pictures. Since the following case of clinical bullous keratitis is, to my knowledge, the most recent to have been studied histologically, it may be considered a link between the experimental and clinical conditions.

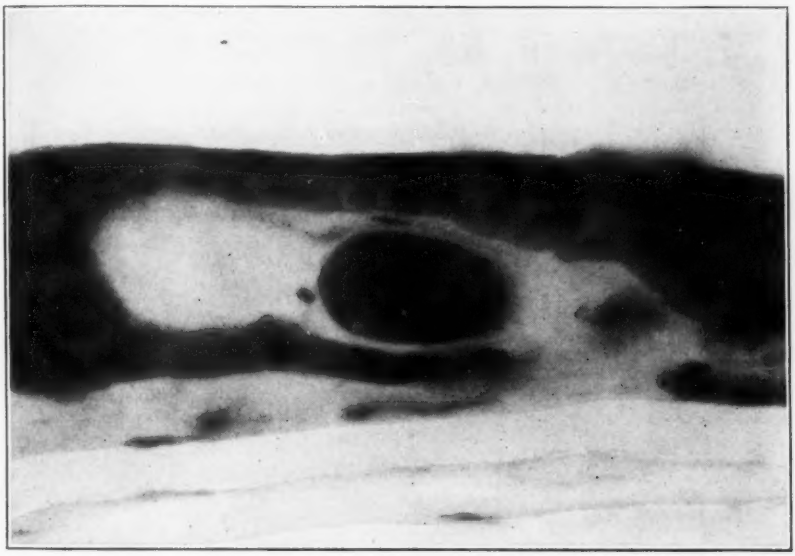

Fig. 9.—Clinical bullous keratitis, showing what appears to be an undermining of the vesicle by basal epithelium. High power magnification.

REPORT OF A CASE

D. K., a 72 year old man with diabetic retinitis, had been examined periodically for several years. On June 8, 1935, the cornea was normal on examination with the slit lamp. There was no elevation of tension to palpation. On July 23 he was seen again, at which time he complained of pain and blurred vision that had begun not more than ten days previously. He was found to have a steamy cornea with a row of small bullae in its center. Two days later the vesiculation had increased and pain was unrelieved. On August 5 he was noted to have marked bullous keratitis on slit lamp examination. On August 7, that is, approximately three weeks after the onset of pain and probably less than that after the onset of bullous keratitis, the eye was removed.

Histologic Examination.—The epithelium varied in thickness. While regions could be found of approximately normal thickness and having the normal number

of layers, the thickness elsewhere was trebled, the epithelium containing several times the normal number of layers. The basal epithelial cells were everywhere swollen. Where they were still in contact with Bowman's membrane they retained their columnar form fairly well. Where they were separated from Bowman's membrane by debris or by tissue (resembling connective tissue) they assumed various shapes. Some were polygonal; others were elongated.

The middle epithelial cells similarly varied from approximately normal shape to marked polymorphism. Polymorphism was especially evident in the areas where the epithelium was thickened. A few intraepithelial vesicles containing granular debris were present, but none of the pearl-like bodies that are found in later stages. The cells lining the intraepithelial vesicles had a columnar shape, resembling basal cells.

Except for some swelliing here and there the surface cells were stratified and appeared most uniformly normal of all the epithelial cells.

Where the epithelium was raised the region between it and Bowman's membrane was occupied either by amorphous debris or by laminated spindle cells. The amorphous debris resembled the cytoplasm of the epithelial cells and contained free-floating, dark-staining bodies that were undoubtedly pyknotic nuclei (fig. 10, top). In some sections disintegrating epithelial cells could be identified in the debris. In other areas the epithelium was elevated for a considerable distance by elongated cells lying in front of Bowman's membrane and coursing parallel to the surface of the cornea (fig. 10, bottom). The nuclei of these cells varied from spindle-shaped nuclei like those of the stroma to an elliptic nuclei not unlike those of the epithelium. This subepithelial membrane contained not more than five layers of cells. No vessels were to be found in it. The membrane in some places merged into the amorphous debris previously mentioned. The basal epithelial cells anterior to the membrane frequently had the normal columnar form.

Bowman's membrane was everywhere intact. The stroma, Descemet's membrane and the endothelium were all normal.

Comment.—In the light of previous observations one may interpret these observations as follows: Some of the basal cells had been so swollen as to burst their walls and disgorge their contents into the subepithelium. Here the mass consisting of loose cytoplasm and nuclei spread out and separated the epithelium. It was then invaded by cells, which most probably came from the epithelium. The invading cells took on a spindle form and arranged themselves parallel to the surface. This was the beginning of Fuchs's "accessory membrane," which in the course of time would presumably have increased in thickness and become vascularized if the eye had not been removed. The process is thus seen to have been one of swelling of epithelial cells followed by their disintegration, with ultimate organization of the debris into a membrane beneath the epithelium.

GENERAL CONSIDERATIONS

Although I have been concerned with corneal changes in reference to bullous keratitis, it is probable that the foregoing observations have a wider significance. Bullous keratitis may represent the late stage of

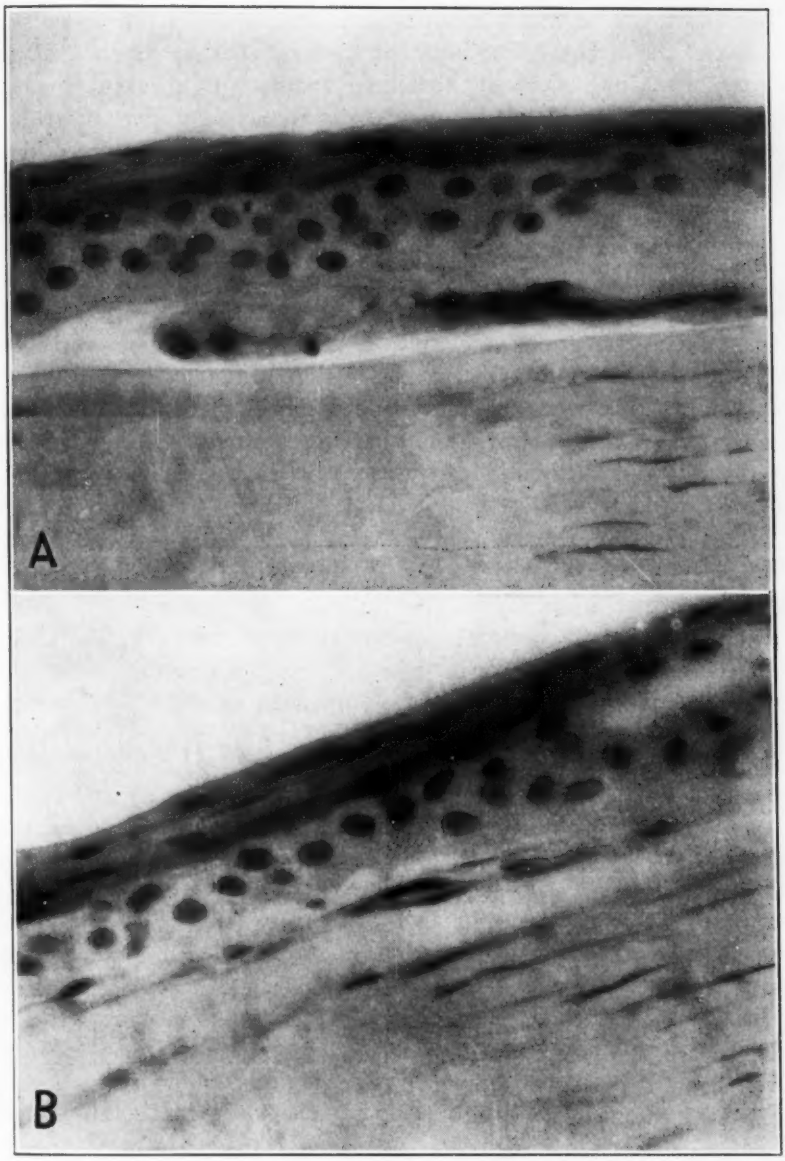

Fig. 10.—Sections of the cornea in the case of bullous keratitis described in the text. High power magnification.

any process in which the epithelium becomes progressively edematous. The origin of the edema fluid appears to be in the tears, and there is no reason to think that it is different in the various types of epithelial edema of the cornea. The fact that edema of tissues generally implies edema of the interstices and that fluid in this type of edema comes of necessity from the blood vessels perhaps accounts for the unwarranted assumption that the source of the fluid is behind the epithelium. As a matter of fact, the direction in which the fluid moves will depend on the gradient of osmotic tension and on the permeability of the barrier. Normally the tears are hypertonic to the corneal fluid and the direction of flow is outward.[32] Under certain experimental conditions and presumably under those clinical conditions which result in epithelial edema, the osmotic pressure of the fluid behind the epithelium—corneal or aqueous—is greater than that of the tears, and fluid is carried into the epithelium. The same process would occur in the skin if its surface were covered with fluid of appropriate osmotic tension. A finger held for fifteen minutes in distilled water will imbibe water and perceptibly increase in size.

This direct interchange between corneal fluid and the environment is perhaps of particular importance for the corneal epithelium, most of which is remote from any blood supply, but the mechanism is after all an old way of controlling salt and water metabolism. In fresh water animals, for instance, in which the surface is permeable by water, fluid will pass from the environment into the tissues or in the reverse direction, depending on the relative osmotic pressures. Indeed, it has been maintained that the excretory organs of certain of these animals act by regulating the osmotic tension of the tissues (Overton[33]). Be that as it may, it would seem that the interchange between cornea and tears is an ancient type of equilibrium and that it is a reversal of the normal balance that results in edema. This reversal might be caused by decreased salt content of the tears, by increased salt content of the cornea or by both. The tears have not been sufficiently investigated in the presence of corneal edema, and as only that part of the tears covering the surface of the cornea has any significance, investigation would be a technically difficult task. At present it would seem more likely that the imbalance results from an increase in the salt content of the cornea or of the aqueous. This appears to be true in the case of

32. Ridley, F.: The Intraocular Pressure and Drainage of the Aqueous Humour, Brit. J. Exper. Path. **11**:217, 1930.

33. Overton, E., cited by Picken, L. E. R.: The Mechanism of Urine Formation in Invertebrates: I. The Excretion Mechanism in Certain Arthropoda, J. Exper. Biol. **13**:309, 1936.

glaucoma, as Fischer [34] has shown for the corneal fluid and Asher [35] for the aqueous. It is immaterial whether the increased salt content is part of the disease *sui generis* or is brought about by the elevated intraocular pressure, but the increase undoubtedly accounts in part for the edema that occurs with glaucoma.

Granted that it is the salt content of the cornea and not that of the tears that is primarily responsible, it is nevertheless possible to produce fluctuations in the epithelial changes by physiologic variations in the tears. Normally the tear film over the surface of the cornea is concentrated by evaporation. This has been emphasized by Fischer,[34] Friedenwald [36] and others, but the most striking demonstration of such a change is Goldman's "salt cataracts" in rats, produced simply by preventing the wink reflex.[37] When evaporation at the surface does not occur the edema becomes considerably worse. I have been able to prove this in cases of bilateral cornea guttata by having the patient wear air-tight goggles with calcium chloride on one side to absorb the moisture. The eye on the side with the calcium chloride improves after an hour, while the other eye gets worse. The absence of evaporation may well account for the usual exacerbation of the edema on waking in the morning. In the case of glaucoma this has been attributed to the increased tension in the morning, but it also occurs with cornea guttata, with which there is no abnormal tension.

Another pertinent observation which several patients with cornea guttata have made is that tearing always aggravates the condition. I interpret this as due to a dilution of the tears or to a washing away of the concentrated film over the surface of the cornea.

RELATED CONDITIONS

The foregoing considerations are general ones which apply as well to the whole subject of edema of the corneal epithelium as to the particular problem of bullous keratitis. Several specific analogies might be mentioned.

The role of osmotic imbalance in cocaine toxicity of the cornea has been suggested in a previous communication.[2] However, at the time

34. Fischer, F. P.: Der Wasserhaushalt des Auges und seiner Teile, Docum. ophth. **1**:79, 1938.

35. Asher, K. W.: Zur Chemie des menschlichen Kammerwassers, Arch. f. Ophth. **107**:247, 1922.

36. Friedenwald, J.: Perspectives in Glaucoma Research, Arch. Ophth. **24**:107 (July) 1940.

37. Goldmann, H., and Rabinowitz, G.: Ueber eine unbekannte, reversible Kataraktform bei jungen Ratten, Klin. Monatsbl. f. Augenh. **81**:771, 1928.

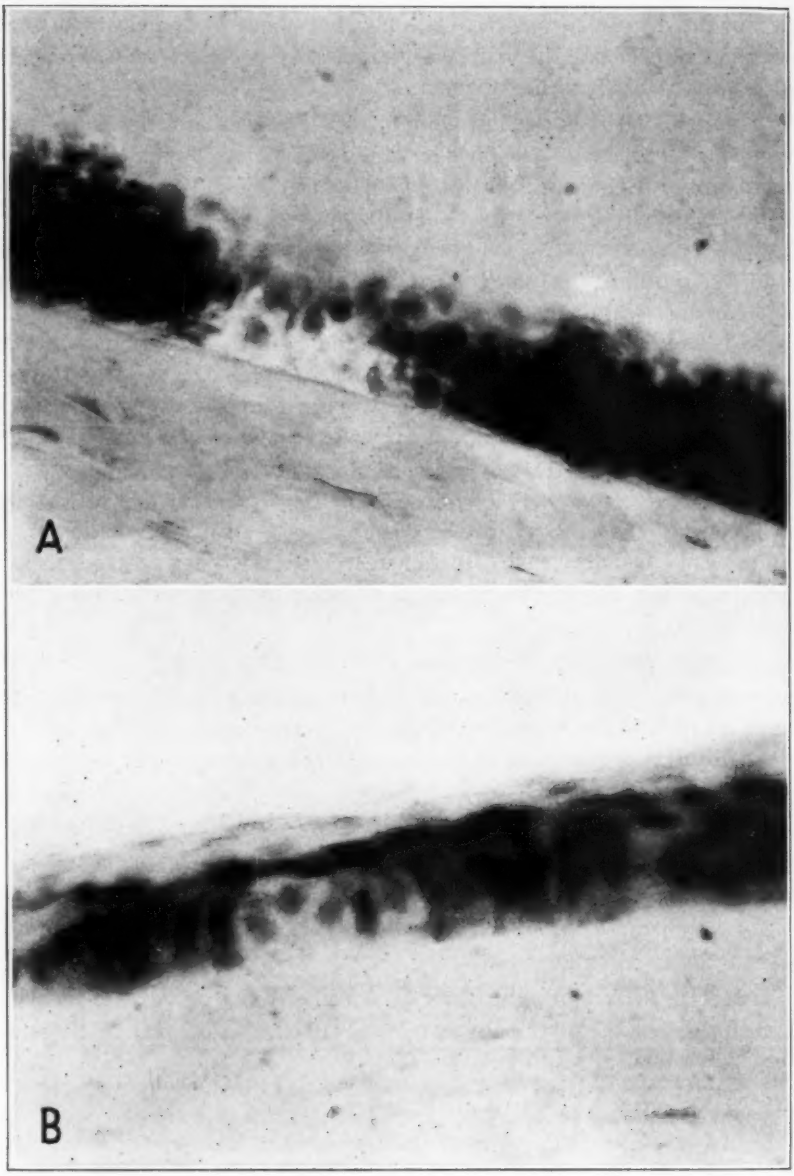

Fig. 11.—*A*, swelling and fragmentation of epithelial cells in a rabbit's cornea produced by 10 per cent cocaine hydrochloride. High power magnification. *B*, irregular swelling and stratification in a rabbit's cornea produced by 10 per cent cocaine hydrochloride. High power magnification.

that communication was written I had not studied the cocainized cornea histologically, and I was unaware of a similar study by Fuchs.[38] Fuchs showed that cocaine hydrochloride produced a swelling of the epithelial cells, frequently with stratification like that described for bullous keratitis. There is, according to Fuchs, not infrequently a vesicular elevation of the epithelium due to cocaine, with conversion of the basal cells into homogeneous-appearing masses containing pyknotic nuclei. I have confirmed these observations and have taken pictures of typical cocainized corneal epithelium to show the similarity of these changes with those in the early stage of experimental bullous keratitis (fig. 11).

Recurring corneal erosion is another condition that is not ordinarily considered in connection with bullous keratitis but has certain features which suggest a basically similar pathogenesis.

Recurring corneal erosion is characterized by epithelial edema with or without bullous formation. The whole epithelium is generally loosened (von Szily[39]). Like bullous keratitis, it is characteristically worse in the morning. So far as I am aware, no tear analysis has been done in cases of recurring corneal erosion, but Curdy[40] reported an interesting case in which healing occurred only after epiphora was relieved by opening a stricture in the lacrimal duct. By applying the outlined explanation of corneal edema to the subject of recurring corneal erosion, one might postulate either one of two things. Those persons who have a relapsing keratitis may have an inherently low osmotic difference between the tears and the cornea, so that any dilution of the tears, as from epiphora, would result in edema. Or one might assume that the cause is basically the same as for cornea guttata and that there has been some endothelial damage which supposedly has resulted in a secondary increase in the osmotic tension of the corneal fluid.

SUMMARY

The theories that have been proposed to explain the development of bullous keratitis are fourfold: 1. Owing to faulty elimination of corneal fluid or to abnormal permeation of aqueous, fluid is passively forced into the epithelium through its posterior surface. 2. The epithelial changes are secondary to dystrophy of the corneal nerves. 3. The changes are secondary to a disease of Bowman's membrane. 4. Owing to an osmotic tension that is greater in the epithelium than in the tears,

38. Fuchs, E.: Ueber Cocain, Wien. klin. Wchnschr. **15**:962, 1902.

39. von Szily, A.: Ueber Disjunction des Hornhautepithels, Arch. f. Ophth. **51**:486, 1900.

40. Curdy, R. J.: Recurrent Traumatic Erosion of the Cornea, Tr. Am. Ophth. Soc. **11**:594, 1908.

fluid is actively attracted into the epithelium through its anterior surface. The experimental production of corneal vesicles and bullae supports the last theory.

The histologic changes associated with experimental bullous keratitis and those observed in the clinical condition are basically similar. There is swelling of the epithelium, which may be diffuse or stratified. The nuclei not infrequently show vacuolation. The entire epithelium is generally loosened and in places separated by vesicles and bullae containing amorphous debris. The changes in the experimental condition make it seem probable that this debris is composed of epithelial cells which, having been swollen to the point of bursting, disgorge their cytoplasm and nuclei beneath the epithelium. This debris is then gradually replaced by layers of spindle cells that resemble those of connective tissue.

Bullous keratitis represents a late stage of epithelial edema, and the mechanism by which tear fluid is attracted into the epithelium in bullous keratitis probably applies in other types of corneal edema as well. Special mention is made of the corneal changes occurring with cocainization and with recurrent corneal erosion.

Edema of the corneal epithelium, which is relatively remote from its blood supply, is not analogous to the usual edema elsewhere in the body. It is presumably dependent more on direct interchange with the environmental fluid (i. e., tears) than with the blood, thus retaining a relatively primitive method of fluid exchange.

RHINOSPORIDIUM SEEBERI INFECTION IN THE EYE

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Mycotic infections of the eye are more often recognized in recent years as knowledge of the various mycoses increases. As many ophthalmologists are unfamiliar with the clinical and pathologic features of a rather rare fungous disease of the mucous membranes of the eye, called rhinosporidiosis, and as the disease in the eye must be more prevalent than the few reported cases would indicate, the discovery of a case in my own practice has been the incentive for a review of the literature on this subject.

CLASSIFICATION OF PARASITE

The parasite was at first thought to be a sporozoon (Seeber,[1] 1900) having analogies with the Polysporea (of Schneider), a subdivision of the Coccidia. Other investigators (Minchen and Fantham,[2] 1905) concluded that Rhinosporidium is an annectent form which shows marked affinities with the typical Neosporidia and also with the simpler Haplosporidia. Beattie[3] (1906) placed Rhinosporidium among the Neosporidia and considered it allied to the Sarcosporidia because of its striated capsule, the framework between the spores and the polar development of the spores. Laveran and Petit[4] (1910) called attention to the resemblance of Rhinosporidium to Ichthyosporidium, which causes an epizootic in trout, and Plehn and Mulsow[5] (1911) were able to show that the latter organism also causes "Taumalkrankheit" in salmon and that it is undoubtedly a Phycomycete and probably allied to the Chytridineae. Ashworth[6] (1923), in his extensive investigation, con-

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Candidate's thesis for admission to the American Ophthalmological Society.

The photomicrographs which accompany this article were taken at the Army Medical Museum.

1. Seeber, G. R.: Un nuevo esporozoaria parasito del hombr. Dos casos encontrados en polipos nasales, Thesis, Universidad Nacional de Buenos Aires, Buenos Aires, Boullosa, 1900.

2. Minchen, E. A., and Fantham, H. B.: Quart. J. Micr. Sc. **49**:521-532, 1905.

3. Beattie, J. M.: J. Path. & Bact. **2**:270-275, 1906.

4. Laveran, A., and Petit, A.: Compt. rend. Acad. d. sc. **102**:421-423, 1910.

5. Plehn, M., and Mulsow, K.: Zentralbl. f. Bakt. (Abt. 1) **59**:63-68, 1911.

6. Ashworth, J. H.: Tr. Roy. Soc., Edinburgh **53**:301-342, 1923.

cluded that Rhinosporidium appears referable to the Phycomycetes, as the thallus is formed of a single cell, and to the Chytridineae, for in this suborder the mycelium is absent or feebly developed and the sexual organs are not generally formed, so that the spores are usually asexual. The spores of most Chytridineae which have been investigated are known to germinate in moisture, and from each a flagellated cell emerges. No flagellated or other active motile stage of Rhinosporidium is known, nor is it to be expected in the case of those spores which, liberated from the sporangium, can settle down in positions in the tissue suitable for their transformation and growth into the trophic stage. Whether it will be possible to observe flagellate stages of the spores in culture remains to be determined. Of the families of the suborder Chytridineae there is one—the Olpidiaceae—in which the cells at the trophic stage are spherical or elliptic, and each such cell becomes transformed into one sporangium only. This is the condition with Rhinosporidium, and hence this genus may be placed provisionally near the Olpidiaceae.

HISTORICAL DATA

The first case of infection with the parasite causing a nasal polyp and a description of the organism were presented by Seeber,[1] in his thesis of Buenos Aires in 1900, lathough he did not name it. It was first named in Belou's "Parasitologia Animal"[7] in 1903, apparently at the suggestion of Wernicke, who, though he had not published any notice of the organism, had referred to it in his lectures. In Belou's book it is called "Coccidium Seeberia—Wernicke 1900." In 1903 O'Kinealy,[8] ignorant of the report from Argentina, reported a case of the parasitic infection, also in the form of a nasal polyp, from India at a meeting of the Laryngological Society of London, but it was not until 1905 that Minchen and Fantham,[2] who were given sections to examine, named the organism Rhinosporidium kinealyi. As Coccidium Seeber-Wernicke (1903) is identical with Rhinosporidium kinealyi (Minchen and Fantham, 1905), the former has the priority and the specific name kinealyi becomes a synonym. The organism is not a Coccidium; the generic title Rhinosporidium was created for it by Minchen and Fantham, and this name, of course, stands. The correct designation of the parasite is Rhinosporidium seeberi (Wernicke, 1903).

Seeber[1] (1900), in the appendix to his thesis, stated that in 1892 Malbran, of Buenos Aires, Argentina, studied sections of a growth labeled "nasal polypus" and that this growth was caused by Rhinosporidium, but no publication was made of this case.

7. Belou, P.: Tratado de parasitología animal, Buenos Aires, 1903, p. 62.
8. O'Kinealy, F.: Proc. Laryng. Soc. London **10**:109-112, 1903.

In 1894 at the Medical College Hospital, Calcutta, India, O'Kinealy saw a Bihari Mohammedan aged 22 suffering from a growth in his left nostril. This growth was removed by O'Kinealy [8] and studied by Vaughan, who made the pathologic report. The data were not published until 1903, when O'Kinealy and Vaughan were still unable to decide whether the parasite should be classified with the Coccidia.

In 1896, in Buenos Aires, Argentina, a growth was removed from the left nostril of an Italian youth, aged 19, who for eighteen years had been a resident of Argentina. Portions of this growth and of a recurrent growth were studied by Seeber (1900 [1] and 1912 [9]), who made his observations known in the thesis which he submitted for the degree of Doctor of Medicine. He considered the parasite to be related to the Coccidia. Belou [7] was the first to publish a name for the organism, though Wernicke had apparently suggested the name in 1900.

In March 1897, Ellett commenced operating on the right nostril of a young farmer who lived near Memphis, Tenn., and who had never been away from the locality. As the growth kept on recurring, he finally sent a specimen (removed in 1902) to Krauss, who later identified it, but nothing was published about this case until Wright [10] reported it, in 1907. This was the first case of rhinosporidiosis recorded in the United States.

Minchen and Fantham [2] (1905) published the results of their observations of material in O'Kinealy's case and, being unaware of Seeber's [1] thesis and Belou's book, named the parasite Rhinosporidium kinealyi.

In 1906, Theiler removed a growth from the nose of a horse in South Africa and had it dispatched to Zschokke [11] (1913), who gave the parasite the name Rhinosporidium equi.

In 1907, Beattie [12] recorded the occurrence of Rhinosporidium in an aural polypus which had been sent to him from India.

Ingram [13] (1910) published an account of a growth on the penis of a Mohammedan from Malabar and of a conjunctival growth.

Minchen [14] (1912) summarized the knowledge of the structure and life history of Rhinosporidium and placed it in the order of Haplosporidia, suborder Polysporulae.

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9. Seeber, G. R.: Rhinosporidium Kinealyi et Rhinosporidium Seeberi, une question de priorité, Buenos Aires, 1912.

10. Wright, J.: New York M. J. **86**:1149-1153, 1907.

11. Zschokke, E.: Arch. f. Tierh. **55**:641-650, 1913.

12. Beattie, J. M.: Brit. M. J. **2**:1402-1403, 1907.

13. Ingram, A. C.: Lancet **2**:726, 1910.

14. Minchen, E. A.: An Introduction to the Study of the Protozoa, London, Edward Arnold, 1912.

Tirumurti [15] (1914) published a summary of the cases observed in India.

Clarke [16] (1915) gave notes on a few examples of Rhinosporidium and in a report of the Institute for Medical Research, Kuala Lumpur, Federated Malay States, recorded examination of a nasal polypus in a male Tamil, a native of Calcutta, India.

Kirkpatrick [17] (1916) recorded from Madras a case of infection of the lacrimal sac. The patient was seen again in 1918 and 1919, and there was then found infection of the inferior meatus of the nose.

Chellia [18] (1918) reported from Colombo an occurrence of nasal polypi. Two patients were Moors but had never been to India, and the third was a Singhalese who had never been out of Ceylon.

Castellani and Chalmers [19] (1919) found Rhinosporidium in a nasal polypus in Ceylon.

Wright [20] in 1922 gave an account of conjunctival growths in 3 Eurasian boys in Madras and noted that in none of them was the nose or the lacrimal sac involved. In the same year [21] he treated one of the growths with 2 per cent aqueous solution of antimony and potassium tartrate dropped in the eye three times daily and found that the growth decreased in size and after three months completely disappeared. In the same year,[21] he recorded a case of infection of the lacrimal sac, the lumen of which was completely filled with polypoid projections from the inner surface. Examination of the nose showed large masses of polypi springing from the inferior and middle turbinates.

On Nov. 6, 1922, Ashworth [6] read his classic monograph in which he traced the history of Rhinosporidium, gave an account of its structure, life history and geographic distribution, stated the results of inoculation and cultural experiments and described the histologic character of the polypus. He came to the conclusion that the organism is not a sporozoon but belongs to the Phycomycetes, suborder Chytridineae, and he provisionally placed it near the Olpidiaceae. He also established its proper name, when occurring in man, as Rhinosporidium seeberi (Wernicke, 1903), and clarified its classification. Finally, he added a

15. Tirumurti, T. S.: Practitioner **93**:704-719, 1914.

16. Clarke, J. J.: Rhizopoid Protozoa, in Protozoa and Disease, London, Baillière, Tindall & Cox, 1915, vol. 4, pp. 107-115; Fourteenth Annual Report of Institute for Medical Research, Federated Malay States, Kuala Lumpur, 1915, p. 71.

17. Kirkpatrick, H.: Ophthalmoscope **14**:477-479, 1916.

18. Chellia, S.: J. Trop. Med. & Hyg. **21**:247, 1918.

19. Castellani, A., and Chalmers, A. J.: Manual of Tropical Medicine, London, Baillière, Tindall & Cox, 1919.

20. Wright, R. E.: Indian M. Gaz. **17**:6, 1922.

21. Wright, R. E.: Indian M. Gaz. **17**:81, 1922.

note on the work of Frey and Hartmann [22] in connection with Rhinosporidium equi (Hartmann, 1921).

Forsyth [23] (1924 and 1933) placed on record the first case of cutaneous infection with this organism.

Tirumurti [24] (1924) reported on the first female patient, whose history of infection went back to 1904.

The first European case, observed in Lombardy, was described by Denti [25] (1925) and by Orlandi [26] (1926).

Parodi [27] (1926) drew attention to the occurrence of Rhinosporidium in Paraguay.

Cordero and Vogelsang [28] (1928) found the infection in a horse in Uruguay.

Cherian and Vasudevan [29] (1929) found a female patient in India with a growth on the fauces and a polypus in the pharynx.

Ayyar [30] (1925 and 1927) recorded tumors due to this organism in the nostrils of bovines in India and in 1932 [31] extended his observations to equines.

Orosa [32] (1929) and Manalang [33] (1929) reported occurrence of the condition in human patients in the Philippine Islands.

Mackenzie [34] (1933) reported its first occurrence in an Englishman, who probably contracted the infection at Kirkee, near Poona, India, where Noronha [35] had special opportunities to make studies of this disease, which is not uncommon in that locality.

O'Flynn and Rudd [36] (1934) in England operated on a Chinese sailor from Canton, but it is not clear where this patient had acquired his infection.

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23. Forsyth, W. L.: Lancet **1**:951, 1924.

24. Tirumurti, T. S.: Lancet **1**:802, 1924.

25. Denti, V.: Boll. d'ocul. **2**:71, 1925.

26. Orlandi, N.: Virchows Arch. f. path. Anat. **262**:314, 1926.

27. Parodi, S. E.: Bol. Inst. clin. quir. **2**:492, 1926.

28. Cordero, E. H., and Vogelsang, E. G.: Bol. Inst. clin. quir. **4**:573, 1928.

29. Cherian, P. V., and Vasudevan, A.: J. Laryng. & Otol. **44**:518-519, 1929.

30. Ayyar, V. K.: (*a*) Mem. Dept. Agric. Industry, Vet. Ser., 1925, vol. 3, no. 6. (*b*) Tr. Seventh Cong. British India & Far East A. Trop. Med. **3**:658-664, 1927.

31. Ayyar, V. K.: Indian J. Vet Sc. & Animal Husbandry **2**:49, 1932.

32. Orosa, S. Y.: J. Philippine Islands M. A. **9**:11, 1929.

33. Manalang, C.: Philippine J. Sc. **28**:437-440, 1929.

34. Mackenzie, K. W.: J. Laryng. & Otol. **48**:107-109, 1933.

35. Noronha, A. J.: J. Trop. Med. **36**:115-118, 1933; Sind M. J. **6**:1-7, 1933; J. Trop. Med. **36**:368, 1933.

36. O'Flynn, J. A., and Rudd, E. T. S.: J. Roy. Nav. M. Serv. **20**:258, 1934.

Allen [37] (1935) reported 5 cases of rhinosporidiosis; 4 of the patients were females.

Allen and Dave [38] (1936) recorded 60 cases of rhinosporidiosis collected during eighteen months in the districts of Raipur and Drug in the Central Provinces, India.

Karunaratne [39] (1936) reported 34 cases which he had investigated personally in Ceylon over a period of thirteen years.

Mandlik [40] (1937) reviewed 48 cases which he had observed during five years. These are of interest because they were collected from areas of infection from water supply which fall in four distinct groups. He presented evidence that rhinosporidiosis is a water-borne disease.

Rao [41] (1938) reported on 18 bovines and 1 pony infected with Rhinosporidium and observed in a period of six years in the Madras Presidency. He cited evidence that the infection is a dust-borne disease.

Anderson and Byrnes [42] (1939) recorded (from North Carolina) the first case of conjunctival infection in the United States.

SITE AND CLINICAL FEATURES OF LESION

In the great majority of cases reported the lesion occurred in the nose. Other sites were the mucous membrane of the eye, the lacrimal sac, the pharynx, the uvula, the faucial pillars, the tonsils, the external auditory meatus, the penis and (in rare cases) the skin of the face and the scalp. The lesion is always in the form of a more or less irregular polypoid growth, single or multiple, red or pinkish red, with minute cream-colored spots (or dots) distributed over the surface, which are the mature sporangia. Some growths are pedunculated, and others, particularly the multiple ones, are sessile. When the growths are multiple and in the form of very tiny nodules, as they sometimes are when they occur in the conjunctiva, the resemblance to granulation tissue is close. The size of the growth varies from tiny pinpoint granulations, such as are often seen in the conjunctiva, to large solitary polypoid masses. The growths are usually friable, soft and vascular and tend to bleed readily on removal.

SEX AND AGE INCIDENCE

The disease is much more prevalent in males (particularly young males) than in females, though the ages in the cases reported ranged

37. Allen, F. R. W. K.: Indian M. Gaz. **70**:76-78, 1935.

38. Allen, F. R. W. K., and Dave, M. L.: Indian M. Gaz. **71**:376-394, 1936.

39. Karunaratne, W. A. E.: J. Path. & Bact. **32**:193-202, 1936.

40. Mandlik, G. S.: Indian M. Gaz. **72**:143-147, 1937.

41. Rao, M. A. N.: Indian J. Vet. Sc. & Animal Husbandry **8** (pt. 3):187-198, 1938.

42. Anderson, W. B., and Byrnes, T. H.: Am. J. Ophth. **22**:1383-1388, 1939.

from 7 to 60 years. The condition does, however, occur in the female, the first case having been reported by Cherian and Vasudevan [29] (1929). The patient was a woman suffering with a pedunculated tumor which completely filled the right side of the nose and extended backward, its major portion hanging behind the soft palate. In all I have found 19 cases recorded in which the patients were females. In reviewing the cases of infection in the eye, I find that in only 3 of the 20 in which the sex was given did such infection occur in the female. Of the total of 18 patients whose ages were given, 12 were under 16 years of age and 6 were over 32.

HISTOPATHOLOGY AND LIFE HISTORY OF THE ORGANISM IN TISSUE

According to Graham [43] (1932), in the earliest trophic stages the parasites are spherical or slightly oval bodies 6 to 9 microns in diameter, with a relatively thick, clearcut, refractile wall, a distinct nucleus and karyosome. As the organism enlarges the cytoplasm becomes heavily granular. When the diameter of some 40 or 50 microns is reached, nuclear division begins. The daughter nuclei repeatedly subdivide, while the entire cyst continues to increase in size. Cytoplasmic division finally occurs at a cyst diameter of about 120 to 140 microns, and there results a sporangium filled with great numbers of spores. They expand and distend the sporangium to a final diameter of 250 to 300 microns. Ashworth [6] has found as many as 16,000 spores in a mature sporangium.

During the middle term of development there appears an annular thickening of a wall about a focus that is destined to become a pore through which the spores will be discharged after maturation. The pore thus established is directed toward the surface of the polyp. The spores lying nearest this pore enlarge and differentiate, while those at the opposite side remain small and immature. The adult spores are round or oval, those of the former shape averaging 7 or 8 microns in diameter and the elongated forms reaching 9 or 10 microns or, in some cases, even 12 to 13 microns, in length. They are outlined by a distinct membrane and contain a nucleus with a prominent karyosome. The cytoplasm is distinct in the earliest recognizable phases but soon becomes vacuolated and then obscured through an increasing development in it of round bodies said by Ashworth [6] to consist of proteinaceous material and called by him spherules. It is these spores which may be seen in the discharge and in great numbers in sections, lying on the epithelial surface, particularly in its cryptlike folds. No other phases than those described here have been observed. As far as is now known, they comprise the entire cycle of development within the human host. It is probable that this cycle is continuously repeated through progress of the spore into the

43. Graham, G. S.: Am. J. Clin. Path. **2**:73-85, 1932.

encysted phase, but it is not known exactly how this takes place. It is not known how the original infection occurs or how autoinoculation at new sites is to be explained.

Ashworth [6] demonstrated that the so-called spore morula is a unit rather than a collection of equipotential spores. He denied the previously accepted opinion that these contained bodies are sporozoites or a cluster of spores contained in a spore morula. He expressed the belief that the spores develop directly into the trophic form. Just where or how this occurs is still a problem. It seems to be assumed that the sporangia mature normally within the subepithelial tissues and discharge their

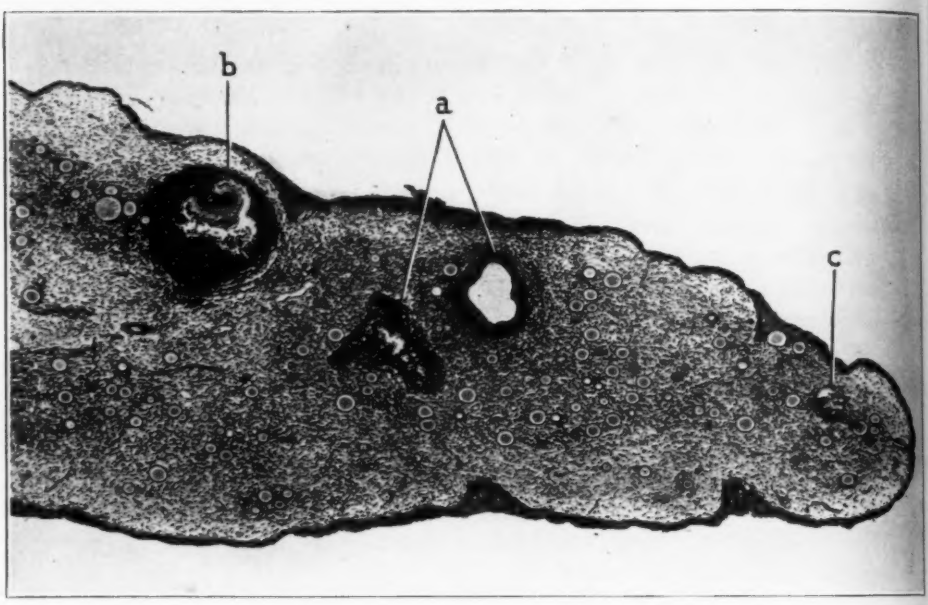

Fig. 1.—Section through the entire thickness of the lesion, showing the granulation tissue reaction and the epithelial changes (× 70). These are of two kinds, thinning over the more active inflammatory areas and proliferation from chronic irritation. There is epithelial invagination (shown at *a*) into the depths of the lesion. Trophic forms of Rhinosporidium are distributed over the entire section; dark central nuclei appear in some. At *b* a ruptured sporangium lies in a small abscess. At *c* a mature sporangium is cut tangentially; its thin capsule is not yet ruptured and no pyogenic reaction is evident. (United States Army Medical Museum negative no. 69489.)

contained spores directly into the stroma of the polyp. In many specimens most of the matured sporangia are found at the surface, where they can discharge the spores externally. Often they lie free within spaces outlined by infoldings of the surface epithelium, and successive stages can be traced in the extrusion of the entire body through the

covering epithelium from the site of maturation in the upper stroma. Such mature sporangia have occurred in nasal discharge (Ashworth [6]). Spontaneous discharge of spores has been seen in some cases, and unopened forms have been made to rupture and discharge by being placed in water. This observation is of interest in connection with the suggestion of observers in India that the infection may be water borne.

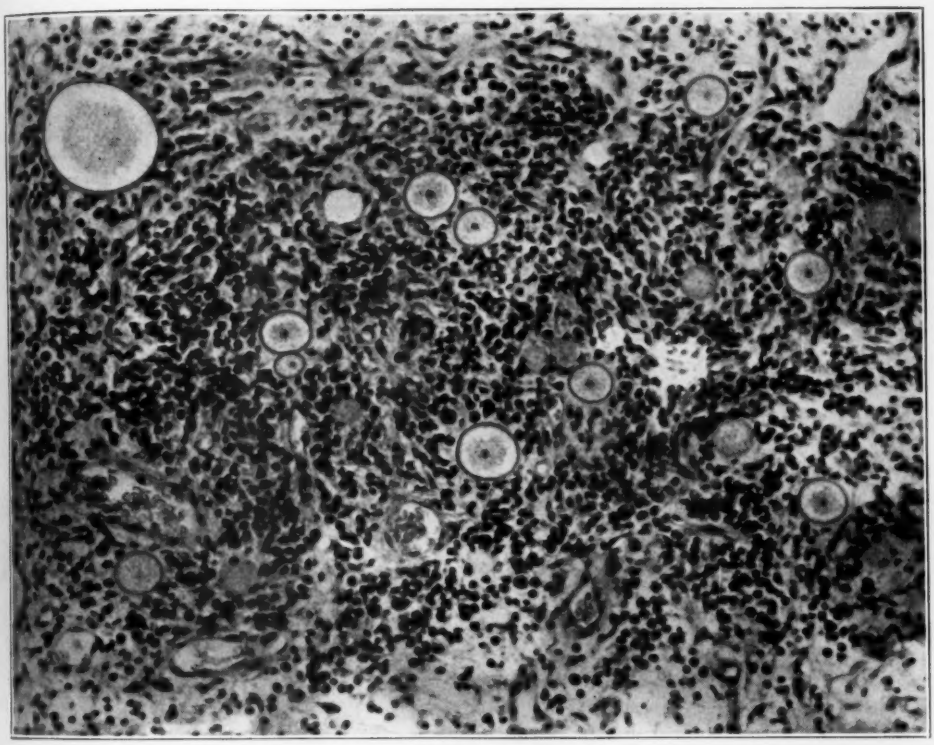

Fig. 2.—Numerous trophic forms with granular protoplasm, distinct central nuclei and a chitinous capsule (× 350). In the left upper corner is a larger parasite with a wider cellulose envelope. Plasma cells and lymphocytes in loose granulation tissue showing numerous engorged capillaries are the characteristic reaction in this stage of the disease. (United States Army Medical Museum negative no. 69488.)

After the spores have been discharged on the surface, reinfection would require penetration of the epithelium. Such an event must be rare. The spore is a body distinctly larger than a red blood cell. There is no evidence that it is ameboid; indeed, its structure suggests rather that it is a rigid body. A search of the available material supports the conclusion of others that intraepithelial forms are rare. In the original

infection and in the occasional instances in which autoinoculation has occurred at new sites, it is probably necessary that the organism find its way in through an open wound of the epithelial covering. It is possible that reinoculation and the initiation of new cycles may take place as the result of leakage of discharging spores into the upper stroma as surface sporangia discharge. When the ripening sporangium enlarges and bulges upward, the overlying epithelium is thinned and may

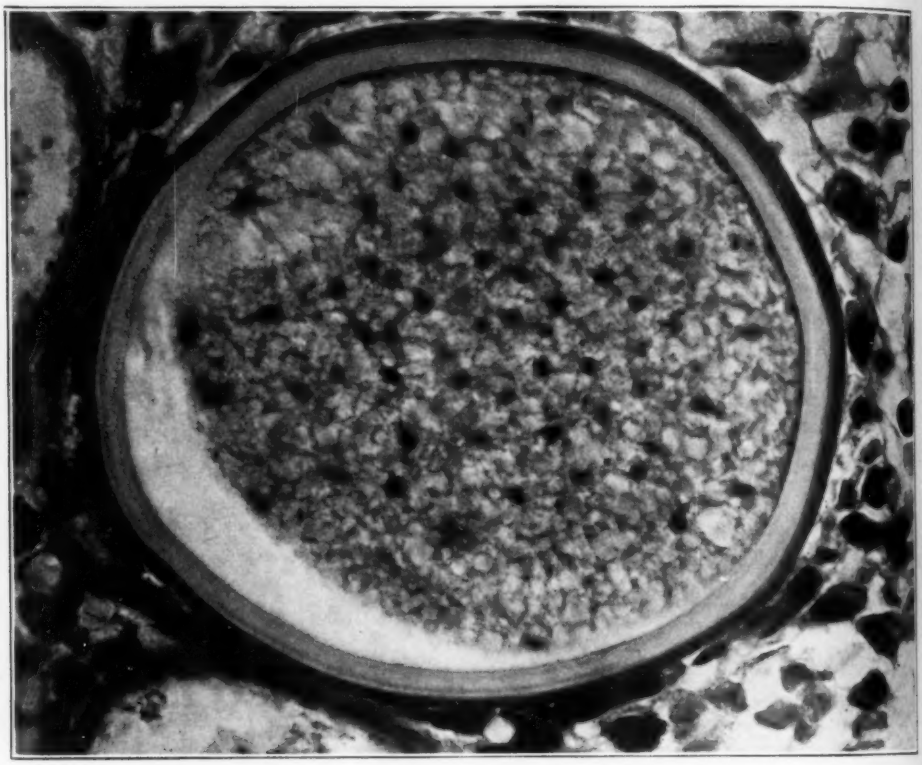

Fig. 3.—Developing parasite with many nuclei in granular cytoplasm (× 1,650). The capsule shows a clear sharply defined cellulose layer within the darker chitinous wall. (United States Army Medical Museum negative no. 68679.)

disappear over an area large enough to allow eventration of the entire body. Again, it may open only in a narrow sector over the site of the pore. As the pore opens and spores are discharged, some may make their way beneath the unseated epithelium, in the space between it and the cyst wall, whence they are carried into the tissue by lymph currents. Or, again, the spores may find lodgment in the stroma within the fresh wound resulting from the expulsion of a discharging cyst.

Finally, new generations may develop directly from spores liberated into the deeper tissue by the rupture of cysts located beneath the surface. This explanation has been accepted by various writers, beginning with Seeber and O'Kinealy, as the normal method of discharge. The subsurface rupture has appeared the most interesting phenomenon exhibited, but it may be interpreted as an accidental occurrence, as contrasted with a normal surface rupture and discharge of sporangium. When it occurs, the spores, possibly accompanied with a little mucinous embedding material, escape directly into the stroma. A microscopic abscess results, starting about the open pore. Unless this abscess is close enough to the surface to open at once on it, liquefaction extends entirely around the sporangium, so that it lies free in a space crowded with neutrophils and large mononucleated phagocytes. Spores, many of them in various stages of disintegration, lie in the space, and the phagocytes are filled with basophilic or acidophilic particles. Occasionally a disintegrating spore can be recognized within a large phagocyte. Eventually the cyst wall spreads wide open and becomes hooped or contorted as though it were an elastic structure that has been relieved of tension. Vaughan, in describing O'Kinealy's specimen, commented on this suggestion of "a certain degree of elasticity." Often the convex surface of the distorted membrane is covered by a thick layer of immature spores, such as may be seen normally at the basal pore of the unruptured sporangium. This suggests that the cyst has turned inside out.

In young cysts that have failed of development or in those that have ruptured, the wall may fragment and particles may be found enclosed within giant cells. Giant cells appear also about the abscess margins to assist in the removal of debris. Resolution of the abscess is followed by contraction of the embedding tissue, and the remaining, central space is filled by the formation of a loose, often edematous scar tissue containing a network of thick collagen fibrils but relatively few active fibroblasts. Within it may be found occasional early trophic forms of the parasite. Such scar areas in various stages of organization may be seen scattered everywhere through the stroma. The cyst wall of the early trophic form was stated by Ashworth [6] and others to be chitinous. In later development there is laid down inside it a thick layer of material "indistinguishable from cellulose." The resulting stratification is often clearly defined in stained specimens. The old suspicion that reseeding of the tissue takes place from the spores liberated within the abscess cavity is believed to be well founded. Some of these spores probably resist the destructive activity of the host's cells and survive to be carried into the marginal tissue or to develop within the scar tissue laid down at the site of the abscess. The adult spore, as it lies within the sporangium or on the epithelial surfaces of the polyp, is outlined by a thin membrane or capsule.

In the trophic forms, from their earliest recognizable stages to their latest stages, the thick wall is the main distinguishing feature of the encysted stage as distinguished from the spore stage. Within the abscess cavity, as occasionally within surface forms, there can be made out thickening of the capsule and increased density of its surface. As was noted by Ashworth,[6] this change is accompanied with a decrease in the size, and finally in the number, of the spherules. While no satisfactory

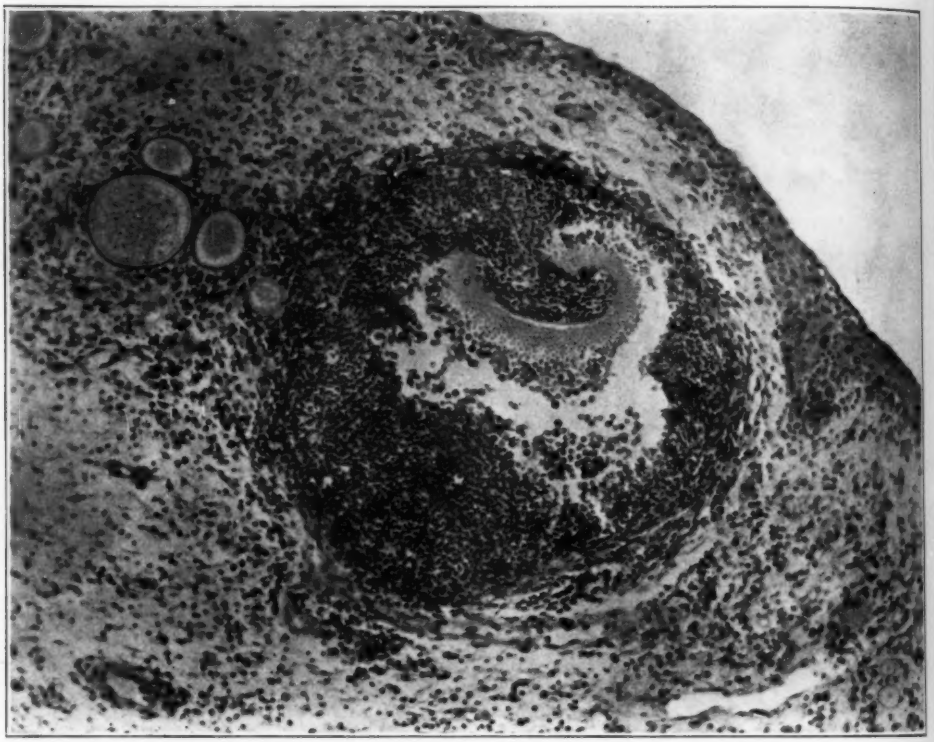

Fig. 4.—Ruptured sporangium lying in a small abscess (× 235). The capsule has looped back by virtue of its elastic character; adjacent to it are crowded pale immature spores with eccentric nuclei. Larger, deeper-staining mature spores cluster about these and lie free in the cavity. The reaction at this stage is that of an acute inflammation; polymorphonuclear leukocytes and numerous eosinophils are present. The epithelium is thinned over the area of the abscess. (United States Army Medical Museum negative no. 68455.)

series of forms has been traced, it appears probable that this transformation proceeds directly to complete disappearance of the spherules, with a medium stage in which there is a well defined cytoplasm that becomes broken into a dispersed granular state as the whole body becomes smaller

and the trophic form is finally developed. These early trophic forms occur within recently developed scar areas and have probably developed here.

OCCURRENCE IN ANIMALS

Although as early as 1913 Zschokke [11] identified an organism which he termed "Rhinosporidium equi" in a nasal tumor in a horse in South Africa, I could find no further reports on the disease in animals until 1926, when Quinlan and de Kock [44] reported 2 cases of nasal growths in mules. In 1927 Ayyar [30b] gave an account of a tumor in a pony, 2 in bullocks and 1 in a cow, in South India. All were nasal growths. In

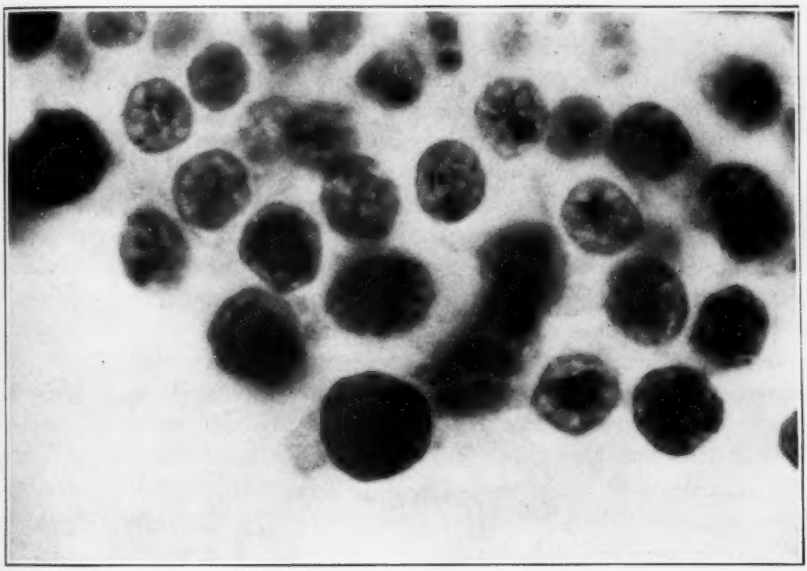

Fig. 5.—Mature spores recently set free show vacuoles, spherules of granular material and central or eccentric nuclei (× 3,000). (United States Medical Museum negative no. 69487.)

1928 Cordero and Vogelsang [28] recorded a case of the disease in a horse in Uruguay, and in 1938 Rao [41] wrote a comprehensive article on the disease in 18 bullocks and a pony in India. In none of the cases was a lesion found in the eye. Only nasal involvements were seen in these animals. The reports are largely from South Africa and India, and Rao [41] expressed the opinion that the disease is much more prevalent in animals than has been recognized.

Rhinosporidium equi, which causes the disease in equines and bovines, is probably identical with Rhinosporidium seeberi, which causes

44. Quinlan, J., and de Kock, G.: South African J. Sc. **23**:589-594, 1926.

the disease in man. Cifferi, Redaelli and Scatizzi [45] (1936) were unable to find any difference between Rhinosporidium seeberi and Rhinosporidium equi after mycologic and histopathologic studies. This adds one more condition to the list of diseases common to man and animals and probably communicable from one to the other.

PROBABLE MODES OF INFECTION

Ashworth [6] referred to direct transmission of the discharged spores from the lesions of rhinsporidiosis as an obvious method of spread from man to man, but such direct transmission has failed in the hands of workers who used laboratory animals for the purpose. There is evidence to show that personal contact even for a number of years does not result in transmission. An example of failure of direct transmission by such personal contact may be mentioned. Kurup [46] (1931) reported the case of a man with lesions in his pharynx who lived with his wife and family for eight years without infecting any of them. Rao [41] (1938) has repeatedly failed to transmit the disease directly with the spores from lesions of diseased animals to healthy bovines or laboratory animals. Hence it would appear that in nature direct infection with large spores from the lesion does not take place. If the discharged spores obtained in the parasitic stage of rhinosporidiosis are not infective, the only alternative is to admit the existence of a saprophytic form of the fungus, whose spores are infective. It is apparent that in the very first case of rhinosporidiosis the infection could have occurred only from a preexisting fungus outside the animal body, which indicates that a saprophytic form exists.

An analysis of the published reports on the disease by workers in India, notably Wright [20] (1922), Kurup [46] (1931), Noronha [35] (1933), Allen and Dave [38] (1936), Mandlik [40] (1937) and Rao [41] (1938), shows that there is a close relation between rhinosporidiosis and agriculture. In a large number of the cases reported the patients belonged to the agricultural class in India. Allen and Dave [38] said that the condition exists in agriculturists even outside India. Rao [41] found that over 95 per cent of the bovines affected were bullocks used for agricultural operations, chiefly plowing. These data support the view that rhinosporidiosis is primarily an occupational disease. It seems clear that the infection is carried either (*a*) by dust or (*b*) by water.

(*a*) *Dust-Borne Infection.*—In the recorded cases of rhinosporidiosis in human beings, over 90 per cent of the patients had lesions in the

45. Cifferi, R.; Redaelli, P., and Scatizzi, I.: Boll. d. Soc. med.-chir., Pavia **50**:723-746, 1936; abstracted, Vet. Bull. **8**:281, 1938.

46. Kurup, P. K.: Indian M. Gaz. **66**:239, 1931.

nose. In the series recorded by Allen and Dave,[38] 68.3 per cent were agriculturists and their children, of whom over 97 per cent had the lesions in the nose. Among bovines and equines, 100 per cent had lesions in the nose. The enormous percentage of infection in the nose in men and in animals connected with agriculture at once suggests that the infective material is dust borne and that it exists on the fields. It is possible that infective material reaches the fields through manure, in which a variety of fungi grow. Both animals and man swallow their nasal discharges, and if spores are present they pass out with the feces in the same way as other spores of fungi pass when they are ingested with food or fodder. These spores are likely to germinate into saprophytic fungi, fructify and liberate spores. If the manure is thrown on fields, the dust containing the spores may settle in the mucus present in the nose and eyes of man and animals and under favorable conditions may attack the nasal and conjunctival membranes, producing lesions.

(*b*) *Water-Borne Infection.*—Mandlik [40] (1937) observed that infection with this organism in Poona and its neighborhood is localized to areas divisible into groups and the infection in these groups is localized to polluted water of certain wells, tanks or infected sections of the river course. He held that the spores, protected by their stout chitinous envelopes, escape with the nasal discharge into the water of tanks or pools when bathers with the lesions of rhinosporidiosis swim in them and that these spores infect later some of the unfortunate persons who swim or dive in such water. In other words, according to Mandlik [40] the infection is direct, and direct transmission with spores has been disproved by experimental evidence. Hence, the water must have become infective in some other way. It has been pointed out that surface washings, and with them spores of the saprophytic form of the fungus, may gain entrance to the more or less stagnant water of tanks, wells, etc., in arable lands or areas of defecation. Mandlik [40] (1937) said that tanks, etc., which he found infective are subject to inundation during rains. Hence it is reasonable to suppose that they may receive infective material from surface washings.

Thus it would appear that infection through dust and water can well be explained if the existence of a saprophytic form of the fungus is admitted. The supposition is not unreasonable, since direct infection with spores from the lesion is not possible.

In a majority of reported cases there was no history of contact with infected persons or animals. The occurrence of autoinoculation is clearly shown by the development of additional polypoid growths in adjoining new sites. There is no evidence of generalized hematogenous dissemination in the reported cases.

CULTURES AND EXPERIMENTAL WORK

Attempts to grow the organism in artificial mediums have been unsuccessful, with doubtful exceptions (that of Rettie, reported by Ashworth [6] [1918], and that of Rao [41] [1938]). Rettie placed spores just discharged from a patient on Sabouraud's medium. After six weeks the medium showed 2 small colonies of organisms. On microscopic examination 1 of these colonies revealed bacteria and numerous specimens of an organism with a vesicular nucleus containing a fairly large karyosome. As the culture proved not to be pure, these cells may have developed from one or more organisms adherent to the surface of the sporangia, but there remains the possibility that they had arisen from the spores of Rhinosporidium. I could find no report of further experiments with culture by Rettie.

Rao [41] (1938), working on the theory that in nature a saprophytic form of fungus probably germinates and thrives in bovine dung, since bovines swallow their nasal discharges and with them the spores of rhinosporidiosis, planted small pieces of growths from bovines or the asci obtained by teasing pieces of growths on sterile dung. The results were encouraging, since it was found possible to obtain the same variety of fungus in a series of experimental cultures, and the best growth was obtained invariably at room temperature.

The cultural experiments were extended to material obtained from human beings, and again a fungus resembling that obtained in cultures of bovine material was obtained every time. Subcultures were made on Sabouraud's medium with dung juice in it, but the best medium for subculture was found to be horse dung agar with gland juice added to it. The growth obtained in culture appeared as a fluffy ball of white wool in about a fortnight. In about six weeks a number of small grapelike bodies appeared on the surface; microscopic examination showed that these were composed of a thick felted mass of mycelia and that no fructification had occurred. The fungus has been sent to experts for identification. The fungus obtained from the culture has been implanted into the punctured hole of the nasal septum in a bull calf, and the result is awaited. Rao stated that he will report on this later.

It has not been possible to transmit the infection to the lower animals. Inoculation experiments have been carried out on monkeys, rabbits, guinea pigs and mice by several workers (Ingram, 1910; Tirumurti, 1914; Wright and Cunningham, 1922; Rettie [cited by Ashworth, 1923]; Orlandi, 1926; Shrewsbury, 1933), all with negative results.

GEOGRAPHIC DISTRIBUTION

The occurrence of rhinosporidiosis covers a wide area, and the cases have been isolated. Most of the patients have been East Indians, with

almost as many from Ceylon and a few from South Africa, the Philippine Islands, North America and South America. A solitary European case has been reported, from Italy.

Ingram [13] (1900) recorded the first case of rhinosporidiosis in the eye, a conjunctival polyp originating in the fornix conjunctivae. There was no growth in the nose or in the nasopharynx. This material [47] was obtained from a case of Kirkpatrick, who published a detailed account of it in 1912 (see table). The patient was a native of India, and the report was from Madras.

It is interesting to note that the cases reported from the United States were observed largely in or near the Mississippi valley region, and none of the patients had ever been out of the United States except 1, who had spent three days in Canada. Three cases, 1 of nasal polypi and 2 of conjunctival infection, have been recognized in the past two years in Houston, Texas; the patients have lived in or near the city all or most of their lives.

TREATMENT

Textbooks on tropical medicine and surgery, when they mention the condition, dismiss the treatment in a few lines and recommend removal of the polypi from the nose by means of a snare. Wright [20] has pointed out that this practice should be condemned as positively dangerous, as it may lead to aggravation of the condition by failure to remove all of the infected tissue and cause multiplication of the polypi by implanting the spores in fresh abrasions made at operation. Excision should be wide, and in a locality where this is difficult cauterization after excision should be resorted to.

Wright [21] has reported disappearance of the infection after the use of 2 per cent aqueous solution of antimony tartrate dropped in the eye thrice daily. Both before and after removal of growths, resort has been had to the use of intravenous injections of neostibosan (Bayer), a pentavalent antimony preparation, in doses of from 0.1 to 0.3 Gm. every day or every other day, and although spores in the secretion disappeared, on discontinuance of the treatment they reappeared. Allen and Dave [38] expressed the opinion that the effect of neostibosan is slow and that a much more powerful drug is needed if simple infections are to be treated medicinally instead of surgically. A trivalent antimony preparation, fuadin (Bayer), has been used by intravenous injection also, but with no success.

47. Elliot, R. H.; Ingram, A. C., and Kirkpatrick, H.: Ophthalmoscope **10**: 428-432, 1912.

Cases of Ocular Rhinosporidiosis Reported in the Literature

Year Reported	Author	Patient's Sex	Patient's Age	Patient's Race	Geographic Location	Original Location in Eye	Site of Recurrence	Comment
1910	Ingram [13]	..	..	.....	India	Fornix; conjunctiva	.....	Reported later by Kirkpatrick (1912)
1912	Elliot, Ingram and Kirkpatrick [47]	M	60	.....	India	Conjunctiva lower lid and mass from inner and lower portion of orbit	.....	Two small growths, one about ¼ inch (0.6 cm.) below outer third of lower lid of right eye; another on inner surface of outer third of same lid; excision of both, one on outer side with portions of conjunctiva adherent to surface and one on inner side far back into orbit; latter operation followed by obstinate hemorrhage
1912	Kirkpatrick, H.: Ophthalmoscope **10**: 430-432, 1912	M	..	Hindu	India	Bulbar conjunctiva	.....	Flat red polypus about 9 by 8 mm., with narrow base attached to upper and outer quadrant of eyeball
1912	Kirkpatrick, H.: Ophthalmoscope **10**: 430-432, 1912	..	..	.....	India	Conjunctiva lower lid	. .....	Red nodule size of pea, much less typical than tumor in foregoing case
1914	Tirumurti	..	..	.....	.....	.....	.....	Published reports of 3 cases previously recorded by Elliot and Ingram and by Kirkpatrick
1916	Kirkpatrick [17]	M	32	.....	India	Lacrimal sac	Beneath operation scar (after one month)	Excision of both original and recurrent tumor; typical cysts in fresh specimen
1922	Wright [20]	M	13	Eurasian	India	Upper and lower palpebral conjunctiva	.....	Growths sprung from short narrow pedicles, spreading out laterally; one divided into small lobes suggesting in color and consistency spleen of small mammal
1922	Wright [20]	M	14	Eurasian	India	Palpebral conjunctiva	.....	Single pancake-like growth sprung from conjunctiva by a narrow pedicle, about 2 mm. above margin of upper lid and 4 mm. from canthus
1922	Wright [20]	M	14	Eurasian	India	Just beneath superficial epithelium of bulbar conjunctiva	.....	Minute growth 2.5 mm. in diameter, without pedicle, projecting slightly above general surface; large single venous trunk leading away from it
1922	Wright [20]	..	..	.....	.....	.....	.....	Case 2 of Wright; history republished with note on result of treatment; polypus of right upper lid cured by instillations of aqueous solution of antimony tartrate

1922	Wright [20]	M	..	.....	India	Lacrimal sac	.....	Red, tense swelling about size of filbert in region of lacrimal sac; boggy, as if filled with granulations; section following extirpation revealed a fibrous wall with polypoid projections from its inner surface completely filling its lumen; polypi seen springing from inferior and middle turbinates of nose
1926	Orlandi [25]	F	56	European	Italy	Conjunctiva	.....	.....
1928	Duggan, J. N.: Brit. J. Ophth. **12**: 526-530, 1928	M	52	.....	India	Bulbar conjunctiva	.....	Excision; semilunar fold probable site of origin
1931	Kurup, R. P.: Proc. All-India Ophth. Soc. **2**: 104-108, 1931	M	12	.....	India	Palpebral conjunctiva	.....	Growth from palpebral conjunctiva of upper eyelid; clear pedicle; resembled a piece of pulmonary tissue; accommodated between the eyelids when lids were in apposition during closure of eye; specimen identified on section as growth due to Rhinosporidium kinealyi (seeberi)
1931	Rao, B. K. N., and Rao, B. V.: Proc. All-India Ophth. Soc. **2**: 109-112, 1931	M	40	.....	India	Palpebral conjunctiva	.....	Polypoid tumor about 6 by 10 by 3 mm., reddish, soft, papilliform, inclined to bleed on pressure, pedunculated; attached to tarsal conjunctiva about middle of tarsus, hanging downward, covering upper cornea and pupil when eye was normally open; tumor mass freely movable without fixation to tarsus and with no induration around its attachment; eye otherwise normal; section of piece of conjunctival tumor showed existence of Rhinosporidium seeberi
1936	Allen and Dave [38]	F	11	.....	India	Palpebral conjunctiva	.....	Treatment declined; when seen again, two months later, nodule had practically disappeared; spores demonstrated in a small roughened area dissected off the conjunctiva
1936	Allen and Dave [38]	M	9	.....	India	Palpebral conjunctiva	.....	Excision; final result unknown
1936	Allen and Dave [38]	M	7	.....	India	Palpebral conjunctiva	.....	Excision; possibly undergoing natural cure
1936	Allen and Dave [38]	F	9	.....	India	Palpebral conjunctiva	.....	Excision; final result unknown
1936	Karunaratne [39]	..	..	.....	Ceylon	Conjunctiva	.....	.....
1936	Karunaratne [39]	..	..	.....	Ceylon	Conjunctiva	.....	.....
1936	Karunaratne [39]	..	..	.....	Ceylon	Lacrimal sac	.....	.....

Cases of Ocular Rhinosporidiosis Reported in the Literature—Continued

Year Reported	Author	Patient's Sex	Patient's Age	Patient's Race	Geographic Location	Original Location in Eye	Site of Recurrence	Comment
1937	Simson, F. W., and Strachan, A. S.: South African J. M. Sc. **2**: 82-88, 1937	..	..	2 Europeans 3 Natives	South Africa	Conjunctiva	.....	One of these is same case reported with recurrence by Kaye
1938	Kaye, H.: Brit. J. Ophth. **22**: 447-455, 1938	M	16	European	South Africa	Palpebral conjunctiva	Limbus (after 2½ years)	Original excrescences, two large polypi in the right eye; one attached to upper fornix, measuring 10 by 10 by 6 mm., and the other attached to lower fornix near outer canthus, measuring 7 by 4 by 4 mm.; recurrence in limbus of same eye as flat reddish polypus encroaching on and causing mild secondary infection of edge of cornea and covered by vascular conjunctiva whose surface was traversed by several large veins
1939	Anderson and Byrnes [42]....	M	12	North American (Negro)	United States	Palpebral conjunctiva	.....	Irregular lobulated pinkish red growth protruding from lower lid near external canthus, definitely pedunculated and attached in fornix by broad base; measured roughly 8 by 4 by 2 mm.; resembled fungoid granulation following the curettement of chalazion; as it was more firm, more vascular and more definitely lobulated than granulations that follow such procedures, specimen was sent to laboratory; diagnosis of Rhinosporidium seeberi made
1939	Griffey [48]	M	10	North American	United States	Bulbar conjunctiva	.....	Nodular solid mass; encapsulated; roughly pear shaped; 12 by 10 by 6 mm.; attached to upper edge of caruncle and scleral conjunctiva by thin stalk 5 to 8 mm. long; mass lying partly in the palpebral fissure so that lids could not be completely closed at inner canthus; excision; specimen sent to Army Medical Museum for section; diagnosis of rhinosporidiosis of scleral conjunctiva made
1940	Elles (this paper).....	M	14	North American	United States	Palpebral conjunctiva	.....	Red polypoid pedunculated mass, attached to conjunctiva of upper lid near outer canthus; 10 by 10 by 2 mm.; pedicle 4 mm. long; under surface of mass could be seen many minute yellowish specks; notch in body of mass where pedicle was attached, giving appearance of a lily pad; growth excised widely at pedicle base and specimen sent to Army Medical Museum for section; diagnosis, rhinosporidiosis of the palpebral conjunctiva

OCULAR RHINOSPORIDIOSIS IN THE UNITED STATES

Anderson and Byrnes,[42] of North Carolina, recorded (1939) the first case of rhinosporidiosis of the eye. The patient was a Negro aged 12 who had noticed a growth on the conjunctiva of the lower eyelid for the past twelve months. During the first six years of his life he had lived on a farm in the eastern part of North Carolina, but for the past six years he had lived in Durham. He had not been associated with any pets, horses, cattle or agricultural pursuits. There was a pinkish red mass protruding from beneath the lower lid, near the external canthus. The growth measured roughly 8 by 4 by 2 mm.; it was definitely pedunculated and was attached in the fornix by a broad base.

Griffey [48] (1939) reported a case of rhinosporidiosis of the bulbar conjunctiva in a white boy aged 10 in Southern Texas. The child lived on a farm and helped take care of horses and cows. The mass was nodular and solid, encapsulated and roughly pear shaped; it measured 12 by 10 by 6 mm. and was attached to the upper edge of the caruncle and the scleral conjunctiva by a thin stalk 5 to 8 mm. long. The mass was lying partly in the palpebral fissure, so that the lids could not be completely closed at the inner canthus.

REPORT OF A CASE

On March 25, 1939, a healthy-looking boy aged 14, of Baytown, Texas, presented himself for examination. About a week previously he had first noticed swelling of the left upper eyelid and a small piece of red tissue projecting below the lid, toward the outer corner. When he lifted the lid a larger tissue mass on a stem fell down. He had had no pain, no discharge and no disturbance in vision. On questioning, the parents admitted having noticed slight thickness and drooping of the child's upper lid for some time. He had had a Shetland pony, which he had fed, curried and cared for for five years and had ridden daily. He had sold the pony about four months previously and had ridden other horses only occasionally since. The pony had had a sinus in its cheek, a short distance from the left side of the mouth, and water came through this when it drank. The pony was dead when the patient was seen.

Examination.—The left upper lid was slightly swollen and thickened in the temporal half. A slight bogginess was felt over this region. Just below the border of the outer half of the upper lid a red mass of tissue with a fluted edge protruded into the palpebral fissure. On eversion of the lid the conjunctiva appeared smooth but was slightly congested near the outer canthus, and projecting from this area, about 8 mm. from the outer canthus and just above the lower edge of the tarsal cartilage, was a short pedicle, attached to which was a red tumor mass. Beneath the surface of the growth were many yellowish white pinpoint dots. The mass measured 10 by 10 by 2 mm. The pedicle was 4 mm. long and 2.5 mm. wide at its base. The surface of the growth was smooth but on close inspection appeared finely granular. There was a slight notch in the body where

48. Griffey, E. W.: Am. J. Ophth. **22**:1389-1390, 1939.

the pedicle was attached. The whole mass had the appearance of a lily pad. There was no evidence of a pathologic process in the nose.

Treatment.—Procaine hydrochloride was injected into the conjunctiva; the pedicle was widely dissected from its conjunctival attachment, and the mass was removed. The patient was seen recently, and there had been no recurrence.

The specimen was sent to the Army Medical Museum. The pathologic report was made by Lt. Col. J. E. Ashe and Capt. Elbert DeCoursey.

Gross examination revealed a disciform piece of translucent encapsulated tissue 11 by 8 by 2 mm.

Microscopic study revealed that in the stroma and occasionally in the conjunctival epithelium there were spores that in some areas numbered as many as thirty-five per low power field. The encapsulated organisms varied from 30 to 200 microns in diameter. Most of the organisms showed a thick, slightly eosinophilic capsule surrounding a foamy granular material whose center often contained an ovum-like structure with a distinct limiting membrane, a pseudogranular protoplasm and a rounded basophilic nuclear body occupying about one third of the diameter of the structure. The larger organisms contained hundreds of endospores, filling the entire interior. The capsules of the large, mature organisms were thicker and somewhat refractile. Two recently ruptured organisms presented abscess-like foci with leukocytes in and around the ruptured organism and a peripheral margin of epithelioid cells. The chronic inflammatory tissue around many of the growing spores was characterized by diffuse edema and an exudate composed chiefly of plasma cells mixed with lymphocytes, large mononuclears, a few eosinophils and polymorphonuclear neutrophils. A rare multinucleated giant cell without associated epithelioid cells was seen. Around the medium-sized and small encapsulated spores the cellular reaction was not intensified, but, since the cells were not numerous around the recently released small forms, it would seem that the rupture of mature organisms had incited the cellular activity. Each escaping endospore would seem to have begun a new cycle of enlargement of an organism to form new endospores. The tissue was vascular, the capillary epithelium often being prominent.

The diagnosis was rhinosporidiosis of the tarsal conjunctiva.

SUMMARY AND CONCLUSIONS

A review of the literature on a rare fungous disease, due to Rhinosporidium seeberi, is given. The naming of the organism is discussed, and its appearance at various sites in the human body and in animals in widely separated geographic districts is traced chronologically from its first recognition until the present time.

The life history of the organism in the tissues and its histopathology, as well as its sites of predilection in the tissues of the body, the clinical features of the resulting lesions and the sex and age incidence, are discussed.

The occurrence of the disease in animals, the probable modes of infection and the results of attempted culture of the organisms and of experimental inoculation are reviewed.

The geographic distribution and the methods of treatment, together with the recording and classifying of all cases of the disease in the eye

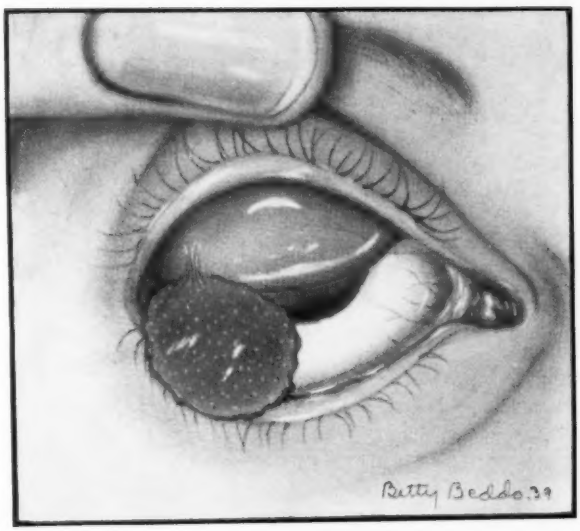

Fig. 6.—Rhinosporidiosis of the palpebral conjunctiva.

UNIV. OF MICH.

that have been reported in the literature and the report of 3 cases of the ocular condition discovered in the United States, 1 of which occurred in my own practice, are described.

It is of interest to note the length of time the lesion has been present before the patient suffers inconvenience enough to seek medical aid; the tendency to recurrence; the length of the intervals before recurrence of the lesion; the size and extent of the tumors compared to the little inflammatory reaction in the tissues and the lack of involvement of neighboring tissue; the absence of systemic complications, and the benign character of the growth.

EXFOLIATION OF THE LENS CAPSULE (GLAUCOMA CAPSULARE)

REPORT OF TWO CASES BEARING ON THE ETIOLOGY, NATURE AND SIGNIFICANCE OF THE CONDITION

RODMAN IRVINE, M.D.
LOS ANGELES

In a previous paper [1] I suggested that study of exfoliation of the lens capsule would lead to a better understanding of the embryology and anatomy of the zonular fibers and their attachment to the lens capsule. A number of questions were outlined, answers to which would give a clearer insight into the nature, etiology and significance of exfoliation.

The 2 unusual cases of exfoliation which I shall report here relate particularly to a number of these original questions:

1. Do the zonular fibers attach themselves to the capsule so as to form a superficial capsule layer, the so-called zonular-lamellar layer, and does this layer extend all the way around the lens?

2. Does pathologic involvement of the most superficial layer of the lens capsule indicate pathologic involvement of the zonule and, since the zonule is supposedly derived from the tertiary vitreous, pathologic involvement of the vitreous?

3. If the zonule is affected when there is pathologic involvement of the most superficial capsule layer, does such involvement indicate altered steadfastness of the lens?

4. Since separation of the zonular-lamellar membrane occurs in cases of "fire cataract," can senile exfoliation of this membrane occur following other forms of irradiation?

5. Does exfoliation per se alter the prognosis of any concomitant pathologic condition, such as glaucoma or cataract?

REPORT OF CASES

CASE 1.—A 77 year old farmer had spontaneous dislocation of the lens of the right eye, of approximately six months' duration. (The left eye showed typical exfoliation of the lens capsule, slight nuclear sclerosis, vision of 20/30 and normal tension.) There were typical scurflike exfoliated flakes on the pupillary margin

From the Section of Ophthalmology of the Department of Surgery, University of Southern California School of Medicine.

1. Irvine, R.: Exfoliation of Lens Capsule (Glaucoma Capsularis): Forty Cases of Exfoliation, Arch. Ophth. **23**:138 (Jan.) 1940.

of the right eye and as marked exfoliation of the capsule as I have ever seen (fig. 1). The lens was dislocated downward, so that the upper edge was in the middle of the pupillary space, exposing to view the upper zonule, which was

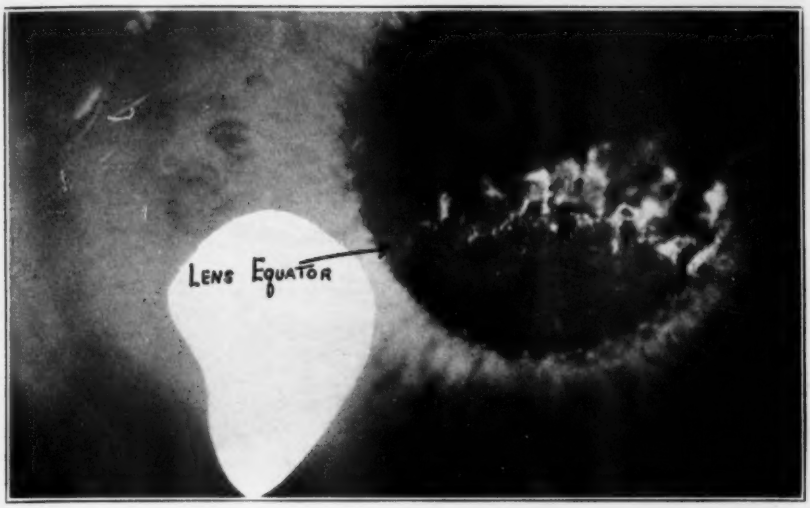

Fig. 1 (case 1).—Slit lamp picture of dislocated lens, showing extreme exfoliation of the capsule of the lens at the equator.

Fig. 2 (case 1).—Slit lamp picture of dislocated lens, showing marked involvement of the zonule in the exfoliating process.

thickened and obviously involved in the exfoliating process (fig. 2). In the resulting picture the zonule appeared to be part of the lens capsule and to be breaking away from it. There were white scurflike shavings leaving the capsule and

streaming along the zonule. The impression from the slit lamp picture was that the zonule was really a ribbed membrane as originally described by Zinn in his "Descriptio Anatomica Oculi Humani" (Göttingen, 1755) [2] and not composed of

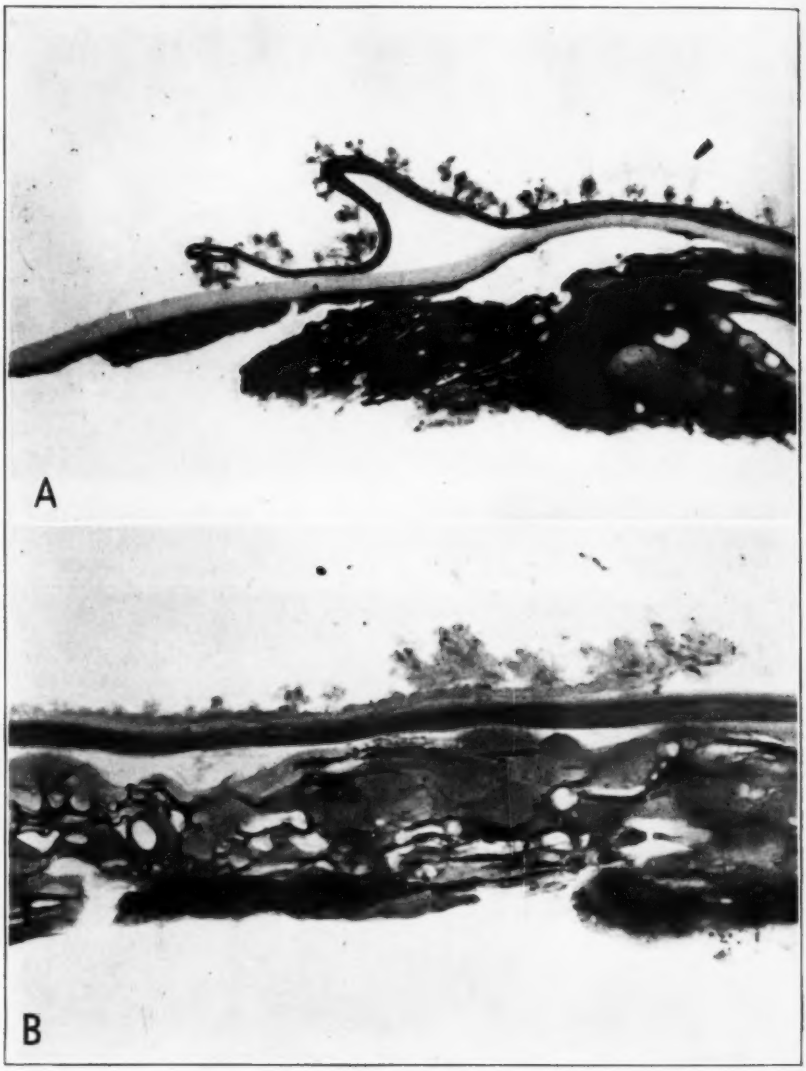

Fig. 3 (case 1).—Photomicrographs of the lens, showing separation and exfoliation of the zonular-lamellar layer of the capsule.

separate fibrillae as is generally depicted. This remarkable appearance of the zonule peeling away in sheetlike formation from the equator could best be produced by

2. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 131.

breaking and subsequent exfoliation of a ribbed membrane encompassing the lens at the equator (figs. 1 and 2).

The tension was moderately elevated in this eye, and a cataract extraction was performed without mishap. The lens floated up to the anterior chamber after

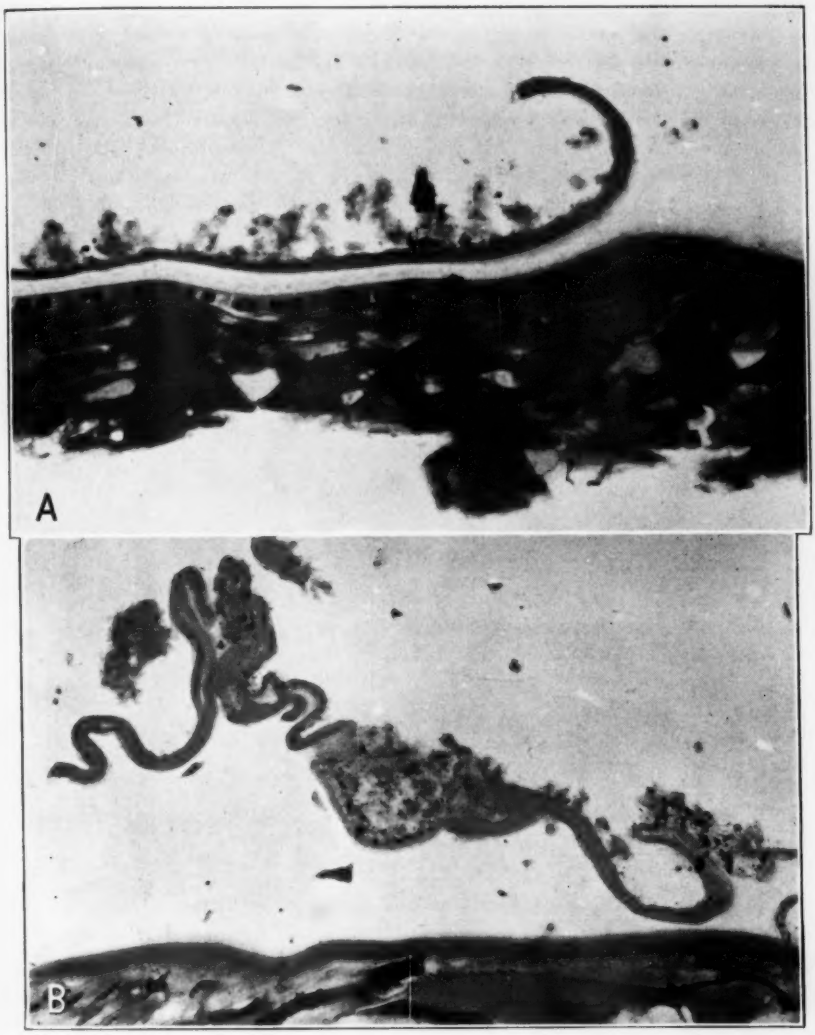

Fig. 4 (case 1).—Photomicrographs of the lens, showing superficial layer of the capsule peeling off in an intact sheet.

the section. A broad basal iridectomy was made. The lens was grasped below with an Arruga forceps in order not to spoil it as a specimen and was tumbled (in its capsule) out of the anterior chamber. There was no loss of vitreous. The subsequent vision was 20/20 corrected; the iris pillars were in good position,

and the face of the vitreous was intact. Removal of the lens controlled the tension for three months, but glaucoma then developed, which did not respond to treatment or to operation. The eye was recently enucleated.

The lens was fixed in Zenker's solution and embedded in celloidin (a pyroxylin preparation) and anteroposterior meridional sections were cut slightly off axis to accentuate the zonular attachments. Some sections were stained with hematoxylin and eosin and others with Mallory's aniline blue. Two capsular layers were easily demonstrable, the more superficial one staining darker. This layer was seen only on the anterior surface of the lens and apparently did not extend around the posterior surface. It was presumably the so-called zonular-lamellar layer of Berger (1882). In these sections exfoliation was seen only on this zonular-lamellar layer (fig. 3). Exfoliating material was readily discernible as

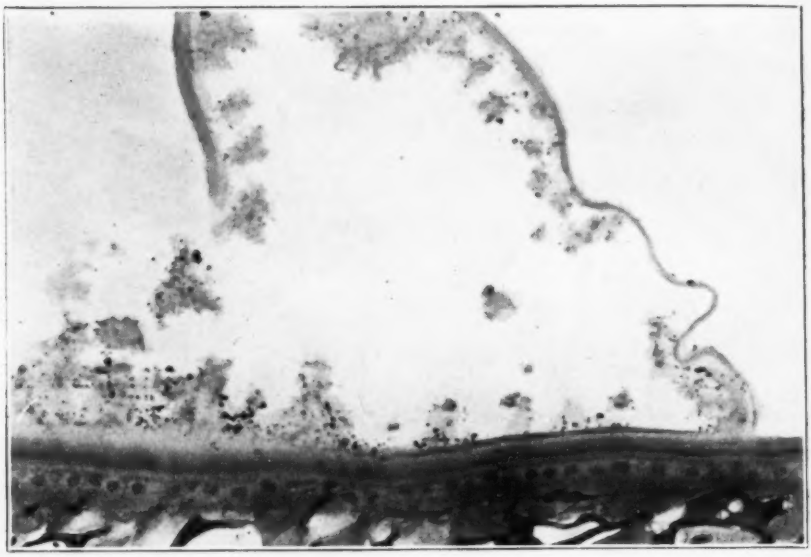

Fig. 5 (case 1).—Photomicrograph of the lens, showing the zonule attached to and becoming part of the zonular-lamellar layer.

amorphous particles coming away from the capsule, especially noticeable near the equator. In some places the superficial layers of the capsule were seen peeling off in an intact sheet (fig. 4). The zonular-lamellar layer itself seemed to be separating from the capsule proper. The zonule attached into and became part of the zonular-lamellar layer (fig. 5), but it cannot be said from these sections that the superficial capsular layer is formed entirely from zonular fibers or that they extend all the way around the lens. The dark-staining layer in question was on the anterior portion of the capsule only, disappearing at the equator. It comprised about one-third the width of the entire anterior part of the capsule. The anterior part of the capsule was about twice as thick as the posterior part.

The parts of the capsule exhibiting the greatest degree of exfoliation were characterized by underlying striations. There was no constant change in the cap-

sular epithelium in these regions. The faint striations appeared to be part of the pathologic picture of exfoliation and may serve as an additional criterion in differentiating true exfoliative capsular particles on the lens from deposition of amorphous coagulum which may be seen in routine microscopic study of sections of eyes with normal anterior segments.

I could find no evidence of exfoliation of the posterior portion of the capsule. Pigment deposits on the capsule were prominent, and in some cases there was fine distribution of pigment granules on the posterior part. The point has been raised previously that the exfoliating material might be a deposition of a coagulum or even of depigmented pigment cells. From some sections one can readily understand how this impression has arisen (fig. 6). Nevertheless, taking the picture as a whole, including the slit lamp appearance, I do not feel that this supposition is correct.

Fig. 6 (case 1).—Photomicrograph of the lens, showing deposition of pigment and depigmented pigment cells coincident with exfoliated capsular remains.

Comment.—In a review of many sections of eyes for the purpose of determining the presence of exfoliation, several eyes with anterior segments known to be normal were seen to have amorphous material on the lens capsule, indistinguishable in appearance from that seen in sections known to show true exfoliation clinically. Apparently vitreous, lens material, aqueous protein and depigmented degenerated cells from the iris may stain similarly to exfoliated capsular material and may be so adherent to the lens capsule as to make the microscopic diagnosis of exfoliation much less certain than the clinical diagnosis.

Trantas [3] was the first to observe exfoliated particles on the suspensory ligament. Later Vogt [4] recorded the same observation, but it remained for Hörven [5] to give it significance. He found the suspensory ligament covered with capsular particles similar to those seen on the pupillary border in every instance in which the zonule could be seen. His study included 128 cases of exfoliation of the capsule seen through an operative coloboma of the iris and an unstated number of excised eyes with exfoliation.

As a result of these investigations Hörven concluded that involvement of the zonule is an integral part of the exfoliation process. He suggested that the ensuing glaucoma might be explained on the basis of altered permeability of the zonular-capsular diaphragm. He noted, however, that his observations did not ascertain whether the zonule fibers themselves were exfoliating or the particles seen on them were the result of deposition as are exfoliating particles on the edge of the iris. In a review of 40 cases of exfoliation I called attention to the presence of a weak and friable zonule associated with the condition, recording 2 cases in which the lenses became dislocated simply after the making of an incision for cataract extraction and 2 instances of tremulous lenses. Sohby-Bey [6] had previously reported 3 instances in his series in which dislocation of lenses had occurred.

In the most recent study of the subject, Gradle and Sugar [7] emphasized the gonioscopic observations in 51 cases of exfoliation. They were the first to note exfoliation of the lamella at the equator, and from their studies they concurred in the belief that involvement of the zonule and the vitreous is an integral part of the syndrome of exfoliation of the lens capsule. Case 1 confirms this belief and furnishes the best evidence to date that the suspensory ligament itself is exfoliating as well as the most superficial layer of the anterior capsule.

CASE 2.—A patient had tumor of both orbits (giant cell lymphocytoma) treated with roentgen therapy. Intraocular examination before treatment revealed no lenticular opacities and no exfoliation. This examination was carried out with the pupils dilated, as it was realized that the necessary amount of radiation therapy might damage the lens. Roentgen therapy was applied as follows (Dr. Kenneth Davis, roentgenologist): The patient received from June 5 to June 18, 1937 a total of 1,050 r to each orbit with an estimated tumor dose of 1,155 r on each

3. Trantas: Arch. d'opht. **46**:482, 1929.

4. Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, Berlin, Julius Springer, 1931, vol. 2.

5. Hörven, E.: Brit. J. Ophth. **21**:625, 1937.

6. Sohby-Bey, M.: Brit. J. Ophth. **16**:65, 1932.

7. Gradle, H., and Sugar, H.: Am. J. Ophth. **23**:982, 1940.

side. The physical factors were as follows: size of field, 10 by 10 cm.; filter, 1 mm. of copper and 1 mm. of aluminum; half value layer, 1.52, and target-skin distance, 50 cm.

A second series was given between July 12 and 18, each orbit receiving 700 r, with the same physical factors specified previously. In addition to this, the left orbit received 802 r between December 20 and 29.

There was complete recession of the tumor, as may be seen from the accompanying pictures (figs. 7 and 8). Approximately two years after irradiation early posterior cortical opacities of the lenses were first noted in both eyes, more pronounced on the left. At that time the pupils were dilated widely again, and

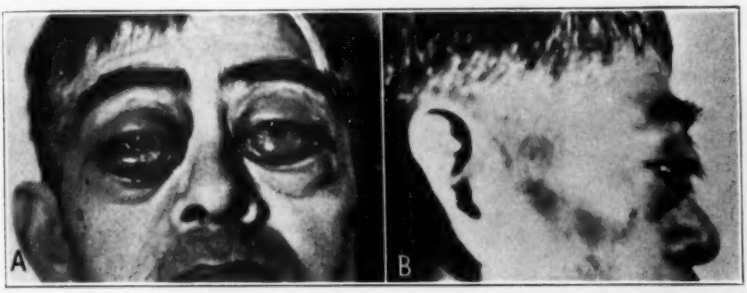

Fig. 7 (case 2).—Patient with giant cell lymphocytoma of orbits before treatment.

Fig. 8 (case 2).—Marked recession of exophthalmos subsequent to roentgen therapy.

marked exfoliation of the lens capsule was seen in the left eye. The exfoliating particles were coming off in sheets (fig. 9). Vision was 20/30 in the right eye and 20/40 in the left eye with correction. There was marked exfoliation in the periphery of the lens and a central pupillary disk. By Jan. 13, 1940 the posterior cortical opacities had increased in both eyes. There was no exfoliation of the capsule of the right eye, but the left eye showed the typical triad of exfoliation, namely, particles on the pupillary edge, a central pupillary disk and a peripheral band of exfoliating particles. The opacities have continued to increase until at

present the vision is 20/100 in the right eye and 5/200 in the left. The right eye still shows no exfoliation, and the exfoliation in the left eye is as described for January 13. There is no evidence of any increase in tension in either eye even on examination with the pupils fully dilated.

I have asked a number of my colleagues associated with tumor clinics to examine (after dilation of the pupils) the lenses of eyes receiving radiation therapy, having in mind the possibility of exfoliation, but as yet no reports have been received. The natural question arising in this case is: If irradiation can produce exfoliation of the lens capsule, why did it occur in the left eye and not in the right? The difference in the amount of radiation therapy to each orbit may afford an explana-

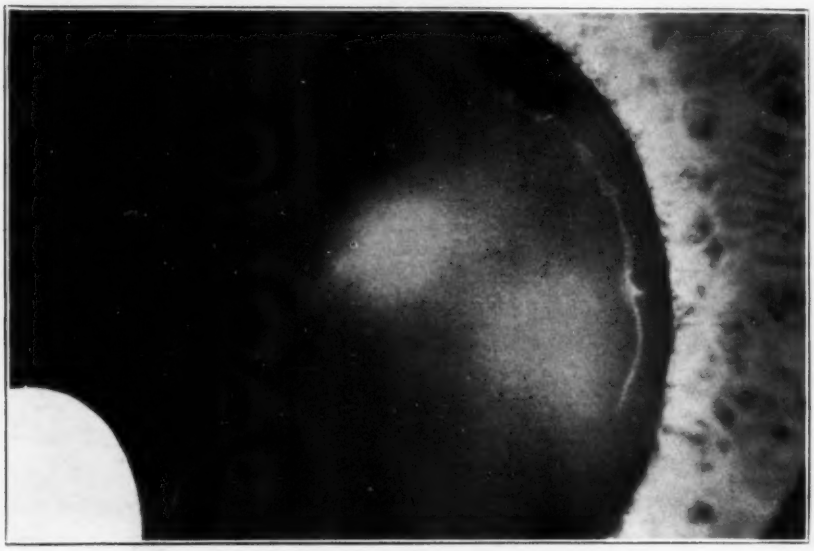

Fig. 9 (case 2).—Slit lamp picture of lens of left eye, showing typical exfoliation of lens capsule two years after roentgen therapy.

tion. It is well known that infra-red irradiation can produce separation of the zonular-lamellar layer as seen with fire cataract, but this is the first case reported in which typical exfoliation of the capsule was seen to occur after roentgen irradiation. Further cases will be needed to confirm a relation.

CONCLUSIONS AND SUMMARY

Two cases of exfoliation of the lens capsule are reported. In the first there was a spontaneously dislocated lens with marked involvement of the zonule in the exfoliating process. This observation and

the microscopic sections of the lens indicate that exfoliation of the lens is a disease of the zonule as well as of the capsule, which suggests that the zonular-lamellar layer of the capsule is similar in structure to the zonule. This case further shows that the zonule is more friable in this condition, altering the steadfastness of the lens. The subsequent course, ending in absolute glaucoma, further indicates that the prognosis of any concomitant condition, such as glaucoma or cataract, is less favorable when exfoliation is present.

In the second case there developed typical exfoliation of the lens capsule after irradiation of the orbit for tumor, a fact suggesting a possible cause of the condition in this case.

BILATERAL GLAUCOMA ASSOCIATED WITH UNILATERAL NAEVUS FLAMMEUS

REPORT OF A CASE

LEON H. EHRLICH, M.D.

NEW YORK

The occurrence of bilateral glaucoma with unilateral naevus flammeus appears to be unique in American literature and has been reported only once abroad. The usual syndrome of glaucoma and naevus flammeus of the skin about the involved eye is still sufficiently uncommon to warrant recording such an association.

This syndrome, to which is frequently attached the names of Schirmer, Sturge and Weber, has been well reviewed in the English language in recent years,[1] and a detailed repetition at this time is unwarranted. A brief description may, however, be appropriate.

In 1860 Schirmer first reported the association of naevus flammeus of the face with buphthalmos. Nineteen years later Sturge presented a case in which there were, in addition to the dermatologic and ocular abnormalities, epileptiform seizures involving the contralateral side of the body, attributed by him to a nevoid condition of the vessels of the brain. Weber (1928), in a case he had first reported six years previously, demonstrated by roentgenographic study of the skull the presence of calcification in the vessels of a meningeal angioma.

To the already mentioned findings—naevus flammeus, usually limited to the distribution of the trigeminal nerve, buphthalmos (or an adult type of glaucoma), epileptiform seizures and calcification of the meningeal vessels—may be added: nevi or dilated vessels of the conjunctiva or of the episclera; dilated vessels or heterochromia of the iris; nevi or angiomas of the choroid; varicosities or tortuosity of the retinal blood vessels, and glaucomatous changes in the optic disk.

The presence of meningeal calcification and epileptiform attacks is the exception rather than the rule, and the syndrome may be considered one of naevus flammeus and glaucoma, either juvenile or adult.

From the service of Dr. Edward Bellamy Gresser at the Beth Israel Hospital.

1. (*a*) Ballantyne, A. J.: Brit. J. Ophth. **14**:481, 1930. (*b*) Granström, K. O.: Acta ophth. **13**:115, 1935. (*c*) O'Brien, C. S., and Porter, W. C.: Glaucoma and Nevus Flammeus, Arch. Ophth. **9**:715 (May) 1933.

REPORT OF CASE

Mrs. A. K., aged 24, born in Turkey, was seen for the first time on May 15, 1940, complaining of blurring of the vision of the right eye and headaches of six months' duration. She was in the ninth month of her first pregnancy. Although the vision of her left eye had been defective since infancy, she had never before sought the attention of an ophthalmologist. There was no history of convulsions or of any other epileptiform manifestations.

Examination revealed her to be healthy appearing, with a naevus flammeus involving essentially the distribution of the first and second divisions of the left trigeminal nerve (see illustration). The buccal mucous membrane of the upper lip, cheek and palate to the left of the midline presented a similar discoloration. The skin on the right side was of normal appearance.

Naevus flammeus and glaucoma.

The vision of the right eye was 20/60; with a + 0.25 D. sph. ⊃ — 1.00 D. cyl., axis 90, it was corrected to 20/20. One tremendously dilated conjunctival vessel ran temporally to the limbus. The cornea measured 13 mm. in its horizontal diameter, and the anterior chamber was somewhat deeper than normal. The corneal sensitivity, the iris and pupillary reactions and the media were physiologically normal. The disk showed a glaucomatous excavation, and in the fundus, around the whole periphery, was a ring of patches of choroidal atrophy ranging up to ½ disk diameter in size, similar to those seen in cases of high myopia. The tension was 45 mm. of mercury (Schiötz).

The vision of the left eye was reduced to light perception and faulty light projection. This eye appeared larger and more prominent than the right. The conjunctival vessels were tortuous and enlarged, although none approached the size of the single dilated vessel seen on the right. The cornea measured 14 mm. in the horizontal diameter, and the anterior chamber was deeper than that of the other eye. Corneal sensitivity was unimpaired. The iris was normal. The pupil

was moderately dilated and reacted sluggishly to light. The media were clear. The disk was atrophic, and glaucomatous excavation, deeper than on the right, was present. Ophthalmoscopic examination otherwise gave negative results. The tension was 55 mm. of mercury (Schiötz).

The next examination was in the hospital on July 31, three days after the birth of the baby. At this time the intraocular tension in the right eye was 32 mm. of mercury, and in the left eye, 65 mm. She was given miotics, which were only temporarily effective in the right eye and partially effective in the left, the tension remaining in the thirties for the right and the high forties for the left.

Study of the visual fields showed a moderate peripheral contraction with enlargement of the blindspot on the right. The left eye was blind except for a small island of vision in the lower temporal field.

Neurologic examination gave negative results, as did general physical examination. Dermatologic examination disclosed no abnormalities except that already noted. Roentgenograms of the skull showed no evidence of calcification of the meningeal vessels.

The Wassermann reaction of the blood was negative. A blood count and a urinalysis revealed no abnormalities.

COMMENT

O'Brien and Porter [1c] in their review of this subject presented 57 cases, including their own. To these may be added 39 cases,[2] some of

2. (*a*) Appelmans: Arch. d'opht. **52**:835, 1935. (*b*) Bengisu, N.: Türk oft. gaz. **2**:253, 1937; abstracted, Am. J. Ophth. **23**:593, 1940. (*c*) Bentsen, K. G.; Dahlsgaard-Nielsen, E., and Möller, H. V.: Acta ophth. **16**:279, 1938. (*d*) Biro, J.: Ztschr. f. Augenh. **88**:80, 1936. (*e*) Chench, A., and Zernitskiy, I.: Vestnik khir. **45**:233, 1936. (*f*) Coppez, in discussion of Joiris and Fanchamps.[2q] (*g*) Corrado, M.: Rassegna ital. d'ottal. **2**:553, 1933. (*h*) Davies, W. S.: Am. J. Ophth. **22**:298, 1939. (*i*) Dehogues, J. L.: Ann. d'ocul. **167**:580, 1930. (*j*) Dunphy, E. B.: Am. J. Ophth. **18**:709, 1935. (*k*) Evans, J. J., and Evans, P. J.: Brit. J. Ophth. **23**:95, 1939. (*l*) Folk, M. L., in discussion on Riser.[2a'] (*m*) Folk, M. L.: Am. J. Ophth. **18**:963, 1935. (*o*) Goldhammer, S.: ibid. **21**:68, 1938. (*p*) Granström.[1b] Imai, N.: Acta Soc. ophth. japon. **36**:114, 1932; abstracted, Arch. Ophth. **12**:112 (July) 1934. (*q*) Joiris, P., and Fanchamps, J.: Bull. Soc. belge d'opht., 1935, no. 70, p. 92. (*r*) Koyama, A.: Chuo-Ganka-Iho **29**:11-16, 1937; abstracted, Zentralbl. f. d. ges. Ophth. **39**:664, 1937. (*s*) Manolesco, D.; Lazaresco, D., and Vintilesco, D.: Rev. d'oto-neuro-opht. **16**:664, 1938. (*t*) de Morsier, G., and Franceschetti, A.: Schweiz. med. Wchnschr. **67**:285, 1937. (*u*) Mullen, C. R.: Buphthalmos with Naevus Flammeus, Arch. Ophth. **19**:831 (May) 1938. (*v*) Mehney, G. H.: Naevus Flammeus Associated with Glaucoma: Report of Case, ibid. **17**:1018 (June) 1937. (*w*) Padovani, S.: Atti d. Cong. d'oftal., 1935, p. 689; abstracted, Zentralbl. f. d. ges. Ophth. **35**:547, 1936. (*x*) Perera, C. A.: Bilateral Buphthalmos Associated with Naevus Flammeus: Report of Case, Arch. Ophth. **14**:626 (Oct.) 1935. (*y*) Pincus, M. H.: Naevus Flammeus Associated with Glaucoma: Report of Case, ibid. **21**:741 (May) 1939. (*z*) Quackenboss, A.: Ann. Ophth. **17**:645, 1908. (*a'*) Riser, R. O.: Am. J. Ophth. **19**:155, 1936. (*b'*) Rumbauer, W.: Klin. Monatsbl. f. Augenh. **101**:866, 1938. (*c'*) Skydsgaard,

the reports consisting only of brief summaries presented at meetings of ophthalmologic societies but included for the sake of completeness. Several cases [3] were not originally published as examples of this syndrome, but the descriptions warrant their inclusion.

Only 1 case was found in which the picture resembled that described in this paper. Clausen [4] described a patient with a glaucomatous excavation of the right disk and buphthalmos of the left eye with a facial nevus on the left side.

In the case published by Koyama [5] there were bilateral buphthalmos, a naevus flammeus on the right side and a naevus pigmentosus on the left side.

The case of Vannas [6] might not strictly be included as an example of the Schirmer-Sturge-Weber syndrome, but it is analogous to that presented here. His patient, a man of 57 years, had a unilateral glaucoma, with a pronounced naevus flammeus of the arm on the same side but not of the face.

The theories of the mechanism of the glaucoma in these cases are many and have been well discussed in other papers.[1] The more plausible ones presuppose telangiectatic changes in the uvea, especially the choroid, and the observations in most of the eyes which have been studied histologically support this contention. The vessel changes provoke disturbances in the intraocular fluid exchange and thereby produce the glaucoma. The appearance of fluorescein in the anterior chamber of the glaucomatous eye but not in the normal eye after its intravenous injection by Tyson [7] and the increased protein content of the aqueous of the involved eye as compared with its fellow as found by Mehney [8] indicate that such disturbances actually exist, at least in some cases.

The response to therapy is not good as a general rule. Miotics are not effective, and surgical intervention is followed by disastrous complications with much greater frequency than in the usual cases of buphthalmos or of chronic simple glaucoma.

H.: Acta ophth. **13**:273, 1935. (*d'*) Steiner, K.: Dermat. Wchnschr. **94**:851, 1932. (*e'*) Stoll, K. L.: Tr. Am. Acad. Ophth. **41**:534, 1936. (*f'*) Vita, A.: Atti d. Cong. d'oftal., 1925, p. 131. (*g'*) Yakovlev, S., and Guthrie, R. H.: Congenital Ectodermoses (Neurocutaneous Syndromes) in Epileptic Patients [cases 9 and 11], Arch. Neurol. & Psychiat. **26**:1145 (Dec.) 1931.

3. Bengisu.[2b] Quackenboss.[2z] Riser.[2a'] Yakovlev and Guthrie.[2g']

4. Clausen, in discussion on Vögele: Klin. Monatsbl. f. Augenh. **81**:393, 1928.

5. Koyama.[2r]

6. Vannas: Finska läk.-sällsk. handl. **76**:399, 1934; cited by Granström.[1b]

7. Tyson, H. H.: Naevus Flammeus of Face and Globe Associated with Glaucoma, Vascular Changes in Iris and Calcified Vascular Growth in Left Occipital Lobe of Brain, with Right Homonymous Hemianopia, Arch. Ophth. **8**: 365 (Sept.) 1932.

8. Mehney.[2v]

SUMMARY

The occurrence of naevus flammeus of the face with glaucoma, usually of the infantile type, has been recorded 96 times. There has been only 1 case in which the ocular involvement was bilateral while the nevus was unilateral. A second case is reported, and mention is made of 2 analogous cases in the literature. Brief mention is made of the symptoms of the syndrome and the cause and treatment of the ocular aspect of it.

FLUORESCENCE MICROSCOPY APPLIED TO OCULAR TISSUES

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AND
EDWARD SINGER, M.D.
BROOKLYN

In histologic sections of the eye it is not possible to identify certain cell components by ordinary microscopic study in visible light. Some of the substances which cannot be seen are of decisive importance for the physiologic function of the eye—for example, vitamin A. Many other cell components and tissue structures can be delineated only after the use of various complicated staining methods which require considerable time. In filtered ultraviolet radiation various substances that have a similar appearance in white light can be readily differentiated owing to their characteristic fluorescence. By utilizing this phenomenon in microscopic examination it is possible to identify a great number of cell components; also it is possible by simple fluorescent staining to differentiate between a variety of cell structures and tissue elements, thereby greatly facilitating the explanation of the function of tissues on the basis of their micromorphologic appearance. This type of microscopic study has been applied to the examination of various other tissues of the body and has given results which would be unattainable with any other method; therefore it is plausible that it could be advantageously applied to the examination of the eye.

TECHNIC

As all previous fluorescence microscopic examinations of the eye have consisted of observations of unstained slides, we had to develop a fluorescent staining technic for this organ. This technic is an adaptation and modification of the methods developed for other tissues by Exner,[1] Haitinger,[2] Keller,[3] Querner [4] and Singer.[5]

From the Department of Ophthalmology, the Long Island College of Medicine.

1. Exner, R.: Ein Beitrag zur Fluorescenzmikroskopie des menschlichen Gehirns, Psychiat.-neurol. Wchnschr. **34**:325, 1932; **36**:291, 1934.

2. Haitinger, M.: Fluorescenzmikroskopie, Leipzig, Akademische Verlagsgesellschaft m. b. h., 1938.

3. Keller, R.: Die Elektrizität in der Zelle, ed. 3, Mährisch-Ostrau, Julius Kittls Nachfolger, 1932.

4. von Querner, F. R.: Der mikroskopische Nachweis von Vitamin A im animalen Gewebe: III. Zur Kenntnis der paraplasmatischen Leberzelleinschlüsse, Klin. Wchnschr. **14**:1213, 1935.

5. Singer, E.: A Microscope for Observation of Fluorescence in Living Tissues, Science **75**:289, 1932.

The tissues can be examined in transmitted or in reflected light. The examination should be carried out in a dark field, as in a bright field the transmitted light from the filter causes light effects that interfere with the fluorescence. The construction of a fluorescence microscope can be seen from figure 1. A detailed description is omitted, as the apparatus was described in a previous publication.[6] For examination in reflected light no sectioning is necessary. For examination in transmitted light frozen sections should be prepared from fresh specimens without any fixation whenever possible. With some practice good frozen sections can be obtained from the various parts of the eye with the

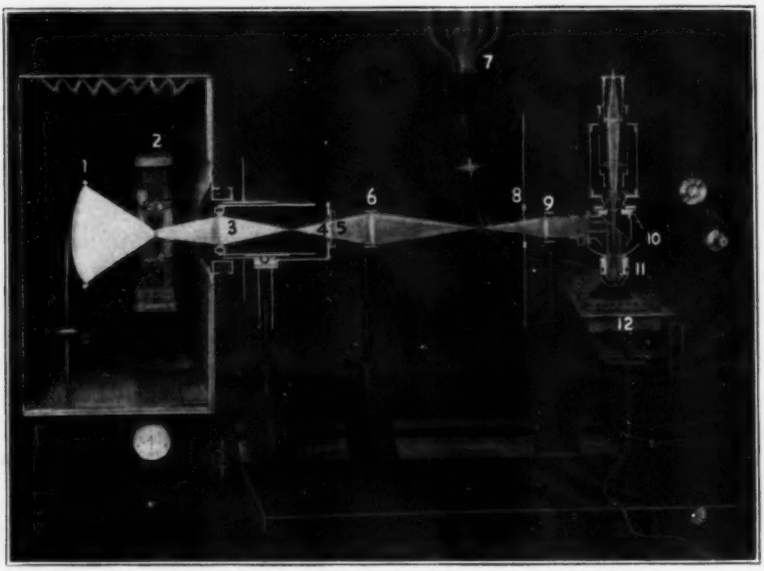

Fig. 1.—Fluorescence microscope. *1*, reflector; *2*, metal arc lamp; *3*, condensing lens; *4*, heat-absorbing filter; *5*, U. V. filter; *6*, condensing lens; *7*, immersion solution; *8*, diaphragm; *9*, condensing lens; *10*, lens illuminator; *11*, focusing device; *12*, mechanical stage.

exceptions of the lens and the retina. These should be fixed before sectioning. Fixation, however, interferes somewhat with the fluorescence. Fixation fluids containing salts of heavy metals or trinitrophenol extinguish the autofluorescence of tissues. Alcohol and acetone dissolve fluorescent substances from the tissues. A 10 per cent con-

6. Singer, E.: A Study of the Morphological Physiology of the Frog's Kidney with the Fluorescence Microscope, Anat. Rec. **55**:37, 1933; Am. J. Anat. **53**:469, 1933.

centration of solution of formaldehyde U. S. P. is most suitable for fixation, as this conserves the fluorescence of tissues well. Tissues should not be kept, however, even in solution of formaldehyde, for more than twenty-four hours, as leaving them in longer causes a fluorescence in tissue elements which do not show any in the fresh state. This fluorescence is most likely caused by dissolution of the fluorescent substances from some tissues and adsorption of the dissolved substances to other tissues.

Color in fluorescent light is due to a colored luminosity of the tissues themselves or to the luminosity of a substance introduced into the tissues. These substances may have color or may be colorless in white light. Thus one is not limited to dyes alone. It is obvious that this color production should not be called staining. Fluorochroming, the term introduced by Haitinger, is a more apt designation for it. In our experiments we used a variety of substances as fluorochromes, such as dyes of the acridine, diphenylamine, xanthene and azo groups, plant extracts, glucosides, alkaloids and vitamins. No relation or similarity exists between many of these substances of various origins, and, although their chemical compositions differ, by their selective properties they can be divided into two groups. We found that one group imparted fluorescence to the nuclei and sometimes to the cytoplasm of the epithelial cells and that the other group never stained the nuclei. The two groups also showed characteristic differences when one examined them for electrical charge. The group which stained the nuclei was negatively charged, and the other was positively charged, against water. The charge could be shown by the way they were adsorbed by filter paper. Members of one group rose into the fibers of the paper with the water in which they were dissolved, and members of the other group became separated from the water as the water rose higher than the dissolved substances. This phenomenon can be explained on the basis of Coehn's law,[7] according to which paper assumes a negative electrical charge when it comes in contact with water. This means that dyes and colorless substances that have fluorescence can be divided into two electrical groups; and again this makes it evident that the electrical charge and staining qualities of the substances show striking similarity and parallelism and that by knowing the electrical potential of a substance one can predict its behavior in the cell. It should be mentioned here, however, that the electrically ambivalent substances will show a charge in the distilled water–filter paper test opposite to that which they will have in tissues. It is obvious that such a simple test as the filter paper

7. Coehn, A.: Ueber ein Gesetz der Elektrizitätsbewegung, Ann. d. Phys. u. Chem. **64**:217, 1898.

adsorption test will give clues which permit far reaching conclusions. It is therefore of interest that a somewhat detailed description of this test should be given.

On dipping the filter paper into the fluorochrome one can distinguish the following zones:

1. An immersion zone, showing the fluorochrome in the same condition as it is in the solution.

2. An anode zone, having a zigzag margin and showing indicator dyes in acid color.

3. A water zone, which appears colorless if the dye is anodic, i. e., negatively charged.

4. A cathode zone, showing the cathodic, i. e., positively charged, dyes in alkali color.

Since the staining qualities and electrical charge of the substances show a striking similarity, one can determine the electrical charge of the substances by this simple test and can divide the substances into the following two groups:

Negatively Charged Substances.—Acridine red, acriflavine (National Aniline & Chemical Company), atabrine (Winthrop), berberine sulfate, geranine G, magdala red, phosphine O, primuline, primuline yellow, rivanol (Winthrop), thiazol yellow, thioflavine S, and all vitamins with the exception of vitamin C are included in this group. (Substances whose firms' names were not given were manufactured by I. G. Farbwerke.)

Positively Charged Substances.—Quinine, uranin and esculin (Eimer and Amend) are included in this group.

Fluorochroming of Ocular Tissues.—In the present study both types of substances were used in concentrated solution (1:1,000 to 1:100,000) for instantaneous staining and in dilute solution (1:100,000 to 1:10,000,000) for sixteen to twenty-four hour staining. The latter staining was used to achieve an even penetration of the fluorochrome.

The usual mounting substances cannot be used, as all of them have a more or less bluish fluorescence and therefore would interfere with the clearness and sharpness of the image. Distilled water is the best mounting material, as all other mounting mediums more or less dissolve some fluorescent substances. Permanent mounts can be made, however, by using liquid petrolatum or nonfluorescent glycerin as a mounting medium. The sections have to be mounted on nonfluorescent and ultraviolet transmitting slides. The slides may have the proper thickness for dark field observation (1.2 mm. or less).

In general the following method can be used for the fluorochroming of ocular tissues:

1. Bathing in fluorochrome.

2. Washing in water that is changed until no further dye goes into the water.

3. Making permanent sections by immersing the sections in a 4 per cent concentration of solution of formaldehyde U. S. P. for ten minutes.

4. Drying the sections on a slide with filter paper.

5. Mounting in liquid petrolatum or, if chlorophyll was used, in glycerin.

A variety of structures which cannot be seen by the usual fluorochroming can be differentiated by diazotization. For this we used the following method:

1. Bathing in watery solution of thioflavine S (1:10,000) for fifteen to twenty seconds or in thiazol yellow (1:1,000) for thirty seconds.

2. Transferring from the aforementioned solution directly into a cup containing the diazotizer (1 to 2 per cent solution of sodium nitrite plus a few drops of concentrated hydrochloric acid), and keeping the sections in this solution for fifteen minutes.

3. Transferring from this solution into bromphenol (1:2,500) for twenty seconds.

5. Washing with distilled water.

6. Mounting in liquid petrolatum.

Material Used.—Human, cat, rabbit, rat and mouse eyes were used.

RESULTS

Differences were noted in the autofluorescence of the eyes in the various species, but no difference was found in the induced fluorescence, the reaction being similar in the eyes of the various species. Many of the findings on autofluorescence confirmed the observations of Böck.[8] Strong luminous bluish fluorescence could be seen radiating from the sclera and Descemet's membrane. The corneal epithelium was somewhat less luminous, and the corneal stroma, the endothelium and Bowman's membrane emitted a weak bluish fluorescence. A slight luminosity radiated from the unpigmented layer of the ciliary epithelium, while the connective tissue of the ciliary body had a considerable bluish fluorescence. The iris appeared in a light bluish fluorescence and its

8. Böck, J.: Untersuchungen des vorderen Bulbusabschnittes mit dem Fluorescenzmikroskop, Ztschr. f. Augenh. **82**:269, 1934.

pigmented part in a brownish fluorescence. The lens was strongly fluorescent and bluish white (fig. 3 *A*).

In the cat's eye the choroid had a strong fluorescence (fig. 6). This fluorescence did not change during illumination. The color of this fluorescence was similar to that of the fluorescence of riboflavin. No proof exists, however, which would support the assumption that riboflavin is present in choroidal tissue. It may be that the tapetum contains some substance of an entirely different nature with a yellowish fluorescence similar to that of riboflavin. The fluorescent tissue may be a characteristic of the cat's eye only, as we found nothing similar in

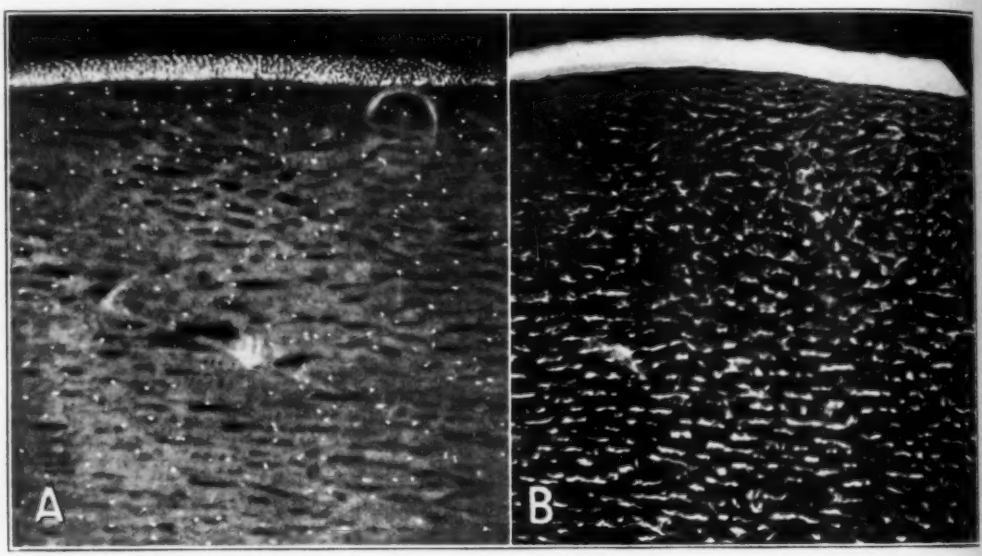

Fig. 2.—*A*, cornea of the cat in fresh frozen section. Rivanol (1:100,000) was the fluorochrome and showed the nuclei in the corneal epithelium and in the corneal bodies. *B*, same tissue as *A*, diazotized. The white band is the epithelium; the irregularly shaped white spots are the corneal bodies, to which thioflavine S imparted a strong fluorescence. The fibers of the cornea, which appear black in the photograph, were reddish brown in the slide, owing to the effect of bromphenol.

the other species studied. We did not find reports concerning this fluorescence in the literature; therefore further investigation is necessary.

The retina has a slightly bluish fluorescence, and all species showed greenish yellow fluorescent dots. The size and number of these dots varied according to the species. These dots disappeared rapidly after illumination with ultraviolet rays. The color and other characteristics

of these dots were described by von Jancsó and von Jancsó,[9] and on the basis of this description they could be identified as vitamin A.

While, as we have stated before, autofluorescence disclosed differences in the eyes of various species, the induced secondary fluorescence did not show any species differences. It delineated, however, the various elements of the tissues.

The negatively charged substances imparted fluorescence to the corneal epithelium. Thioflavine S stained the entire epithelium, while other substances stained the nuclei only. The most selective staining could be accomplished by rivanol. With this substance, if the timing was correct, not the slightest tinge of its fluorescence was perceptible in any part of the tissue outside the nuclei. Rivanol showed up the nuclei in the corpuscles of the corneal stroma also. The fluorochroming with atabrine had the closest similarity to that with rivanol. Other substances imparted fluorescence in a slight degree to the cytoplasm also, which made the appearance of the tissues similar to that of tissues stained with eosin in ordinary microscopic procedures. Diazotization showed the stroma stained with bromphenol, but the corneal cell corpuscles in it did not take up bromphenol and appeared in the fluorescent color of thioflavine S (fig. 2). When the sections were treated with acetic acid no fluorochroming could be accomplished with any of the negatively charged substances.

The positively charged substances showed fluorescence in the stroma of the cornea. This staining, however, was never very distinct and did not show any differentiation. Other parts of the eye also did not show any distinct fluorescence with any of the positively charged substances, which is contrary to our observations in the living eye.

The sclera showed a different hue of fluorescence from the cornea when fluorochromed with the same substances. This revealed a sharp boundary line between the sclera and the cornea. The fibers of the sclera almost always became somewhat fluorescent with fluorochromes which imparted fluorescence only to the nuclei of the cornea. The nuclei were sharply outlined, however, having the shape of spear heads. When diazotized the fibers took the color of bromphenol just as did the fibers of the cornea, and the sclera appeared homogeneously stained with bromphenol (figs. 3 *B*, 3 *C* and 4).

The stroma of the iris did not take any of the negatively charged substances. After fluorochroming with rivanol all nuclei became fluorescent. Thioflavine S imparted fluorescence to the sphincter muscle only.

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9. von Jancsó, N., and von Jancsó, H.: Fluorescenzmikroskopische Beobachtungen der reversiblen Vitamin A Bildung in der Netzhaut während des Sehaktes, Biochem. Ztschr. **287**:289, 1936.

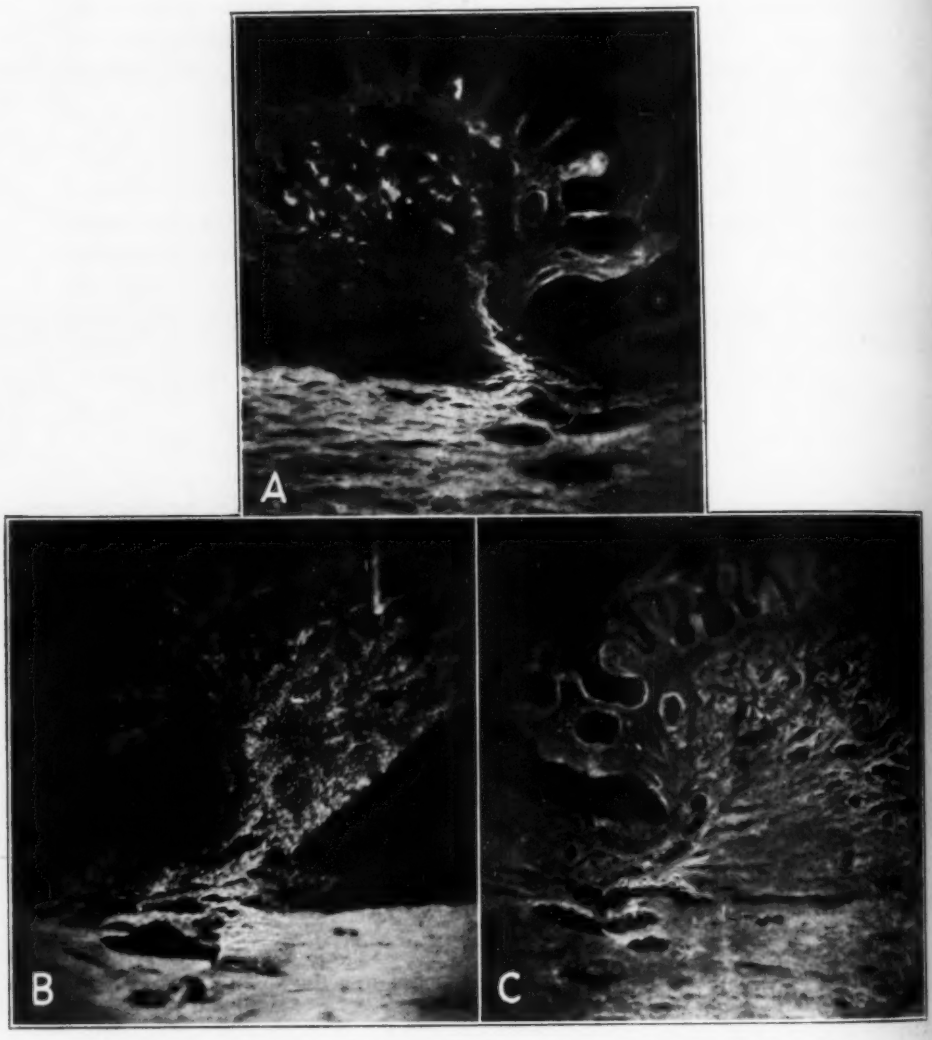

Fig. 3.—*A*, chamber angle of man in frozen section, fixed in solution of formaldehyde. The tissue is autofluorescent. Note the strongly fluorescent sclera. In the ciliary body the ciliary muscle, the connective tissue and Bruch's membrane also appear fluorescent. *B*, same tissue, fluorochromed with rivanol (1 : 100,000). All the nuclei appear sharply outlined. *C*, same tissue, fluorochromed with thioflavine S (1 : 1,000). The entire ciliary body and the sclera appear in the picture sharply outlined, owing to fluorescence imparted by thioflavine S. On some of the ciliary processes one can notice a higher luminosity in the epithelium than in other tissues. It is noteworthy that the pigment epithelium is also fluorescent.

All negatively charged substances imparted fluorescence to the ciliary body. The entire epithelium and the stroma were fluorescent with thioflavine S. With higher magnification one could discern that the

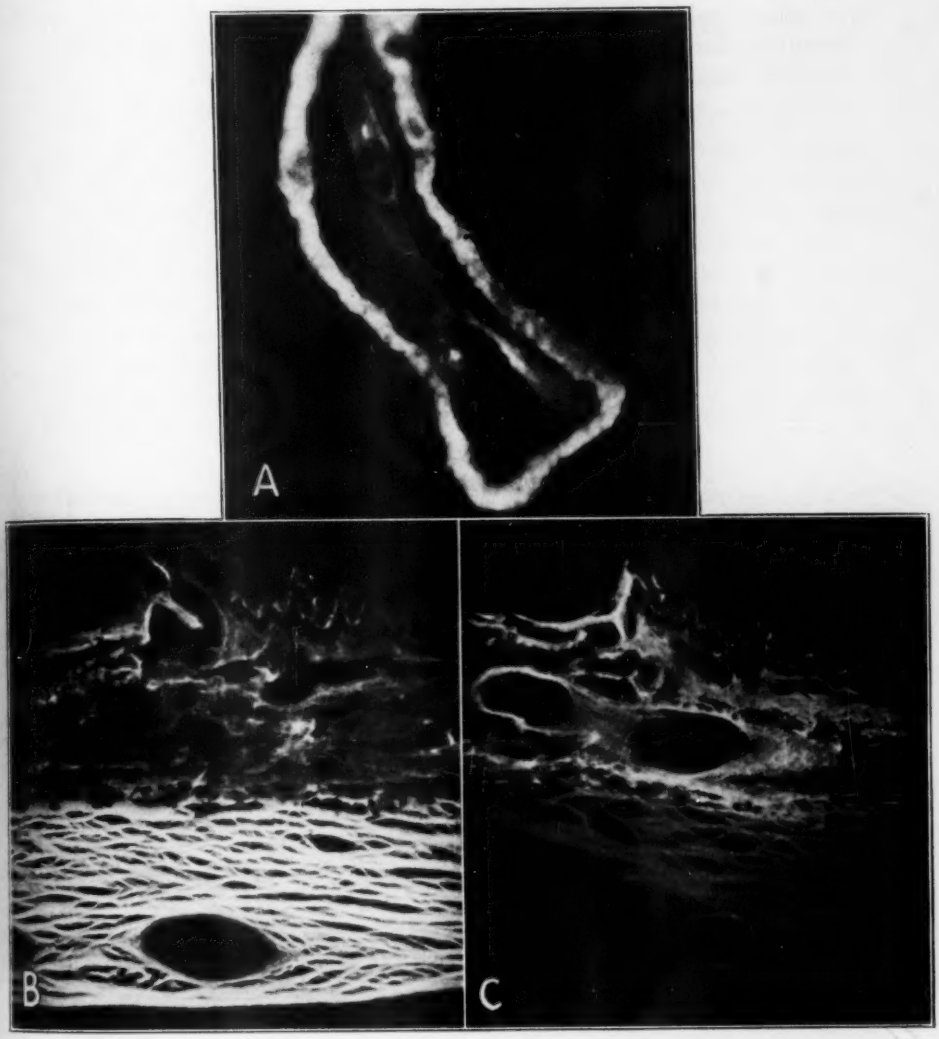

Fig. 4.—*A*, same tissue as in figure 3 *A*, diazotized. The magnification is higher, showing one ciliary process. The ciliary epithelium is highly luminous. The endothelium of vessels appears also, but somewhat less, luminous. *B*, chamber angle of the cat, fluorochromed with thioflavine S (1 : 1,000,000). The appearance is similar to that of the tissue shown in figure 3 *C*, but here the pigment epithelium is not fluorescent and therefore appears as a black band. *C*, same tissue, diazotized. The ciliary body retained thioflavine S, and therefore in the photograph it appears white. The sclera is stained with bromphenol and appears dark in the photograph.

epithelium showed a much stronger fluorescence than the stroma (figs. 3 *B*, 3 *C* and 4). In the cat the pigment layer of the epithelium never became fluorescent, while in man this layer of the epithelium could be made fluorescent. Diazotization did not influence the appearance of the ciliary body, which did not take the bromphenol but appeared as though thioflavine S alone had been applied (fig. 4 *C*). Rivanol stained all nuclei selectively. The number of nuclei in this part of the eye is so great that the entire ciliary body appeared thickly dotted (fig. 3 *B*).

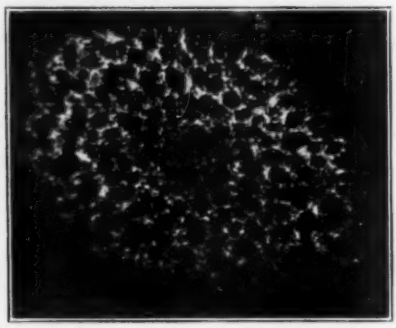

Fig. 5.—Optic nerve of the cat in fresh frozen section. Rivanol (1 : 100,000) was used. All the nuclei are selectively stained and appear very sharply outlined.

Fig. 6.—Choroid and optic nerve of the cat in fresh frozen section. The tissue is autofluorescent. Note the strongly fluorescent choroid, which appears as a white band in the photograph.

The nuclei in the choroid membrane could be made visible with rivanol and berberine sulfate. Rivanol and atabrine imparted fluorescence to the nuclei of the retina. No structural picture could be discerned in the lens with any of the fluorochromes. The lens always appeared diffusely fluorescent, without any differentiation. In the optic nerve all nuclei of its septums and pia could be made selectively fluorescent by rivanol (fig. 5), while thioflavine S stained the nerve fibers.

Frozen sections of the various parts of the eye when fluorochromed with positively charged substances showed only diffuse fluorescence, and there was never any cell differentiation visible with these fluorochromes. This is contrary to the observations on the living eye and also to the observations on other tissues (Singer [10]). Further experiments are necessary to elucidate this phenomenon.

CONCLUSIONS

Two fluorescent colors, bluish white and greenish yellow, appeared in the unstained microscopic sections of the eyes of several animals and of man. In the cat's eye there was a third color, yellow. The strong bluish fluorescence was caused by a substance contained in the tissues, which diffused into glycerin after the tissues had been kept in it for a few weeks. This fluorescence was not decreased after exposure to ultraviolet radiation. No attempt had been made to identify this substance, and no observations have been made on its behavior under various pathologic conditions. It is noteworthy, however, that we found this bluish autofluorescence strongest in the outside layers of the eyeball, i. e., in Descemet's membrane, in the lens and in the sclera. This quality of the outside layer may indicate that ultraviolet rays, if they reach behind this layer, reach the retina in substantially reduced intensity, as fluorescence results from the transformation of short ultraviolet rays into longer ones. It may well be that fluorescence of these tissues has a protective action by withholding the ultraviolet rays from the deeper layers at the same time that it permits the passage of visible rays.

The greenish yellow fluorescence which was found in the retina rapidly lost its intensity by illumination. The substance having this fluorescence was apparently vitamin A. It seemed stored in the retina in a way similar to the storage of artificially introduced fluorochromes, and what is said in the following paragraph concerning fluorescence induced by fluorochromes seems to apply also to that induced by vitamin A.

The secondary, or induced, fluorescence showed structural details only when negatively charged substances were applied. Although these substances had in many ways similar staining qualities they also showed remarkable individual differences. Those negatively charged substances that made the nuclei fluorescent imparted this fluorescence to all nuclei, but, contrary to the staining in visible light, negatively charged substances that imparted fluorescence to the cytoplasm and fibers did not

10. Singer, E.: Observations on the Living Eye with the Fluorescence Microscope, to be published; Histological Aspects of the Function of the Malpighian Body in the Living Frog's Kidney, Based on Studies with the Fluorescence Microscope, Anat. Rec. **66**:343, 1936.

make fluorescent all cells of the various tissues of the eye. The corneal epithelium, the ciliary body and the posterior part of the iris are the tissues that were stained with negatively charged substances. All of these tissues have a positive electrical charge against blood, according to Fischer,[11] who measured their static potentials. The lens and the vitreous body were also found by him to belong to this group. On these two tissues, however, our experiments were not conclusive (fig. 7).

From potential measurements on other tissues it is known that all nuclei of living cells have a positive charge and that the nuclei retain this charge in fresh frozen sections. It is further known from these experiments that if the cytoplasm has a positive charge this charge is

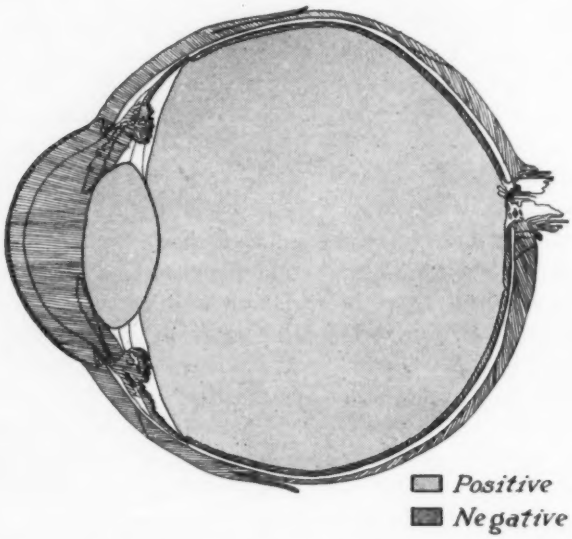

Fig. 7.—Schematic drawing of the eye, showing the electrical charge of the various layers.

imparted to it by a large number of positively charged granules, which overbalance the basically negatively charged protoplasm. In accordance with these findings, the negatively charged substances must have made fluorescent not the cytoplasm but some granules contained in it. Those vitamins that have a negative charge must be stored in these granules. Up to the present time only one, vitamin A, has apparently been identified. In accordance with our results other vitamins can be expected to be localized in the positively charged tissues of the eye.

11. Fischer, F. P.: Vitalfärbungen am Auge, Kolloidchem. Beihefte **28**:333, 1929.

While one might speculate on the possibilities of fluorescense microscopy as applied to the study of pathologic material, no such investigations have as yet been undertaken. Such studies would obviously have to precede any attempt at clinical application. Perhaps the basic principles as set forth in this communication can be applied to studies of ocular physiology.

It is conceivable that recognition of altered electrical potentials with modification of physiologic processes in health and in disease will extend the usefulness of fluorescence microscopy as it applies not only to ocular tissues per se but to the living human eye.

SUMMARY

The fluorescence of frozen fresh ocular tissues has been examined in a dark field.

A strong bluish autofluorescence has been observed in Descemet's membrane, the lens and the sclera. The other tissues showed fluorescence to a considerably lesser degree.

Fluorescence could be imparted to the nonfluorescent tissues and the color of the fluorescent tissues could be changed by fluorochroming, which is a process similar to histologic staining. Fluorochroming consists in application of greatly diluted, highly fluorescent compounds, dyes as well as colorless substances, to the tissues.

All substances used for fluorochroming in the present investigation were classified according to their electrical charge because, irrespective of their chemical characteristics, similar results were obtained with all members of the negatively charged group, and the positively charged substances always showed diffuse staining without any cell differentiation.

The negatively charged substances imparted fluorescence to the corneal epithelium, to the ciliary body and to the optic nerve. Among these substances, rivanol stained the nuclei selectively and thioflavine S stained the entire tissue.

Small droplets of vitamin A, which has a strong greenish yellow fluorescence, could be identified in the retina.

In the choroid of the cat a strong yellow fluorescent substance was seen. The fluorescence of this substance was uninfluenced by exposure to ultraviolet rays.

Dr. Arnold de Veer assisted in interpretation of the histologic slides.

OPTIC NEURITIS CAUSED BY A COAL TAR HAIR DYE

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The presenting symptom of visual disturbances in a patient in whom the sole objective finding is bilateral optic neuritis places before the physician a difficult problem in differential diagnosis. To attribute such a condition to toxic and general systemic causes [1] and overlook the possibility of an expanding intracranial lesion as the etiologic factor may mean to sacrifice precious time during which surgical intervention may save vision or life itself. To overlook a possible toxic cause may lead to unnecessary operative procedures.

REPORT OF A CASE

History.—L. W., a married white woman aged 37, entered the hospital on May 7, 1940, complaining of blurred vision of four weeks' duration. She had been in excellent health until four weeks before admission, when after a long automobile ride in misty weather she returned home greatly fatigued and suffering from frontal headache. She also noted a feeling of "heat" and pain in the eyeballs. The headache was relieved by acetylsalicylic acid. That night she yawned incessantly before falling asleep. The next morning she awoke with severe sharp pain in both eyes and in the region of the temples. She consulted an ophthalmologist, who told her that she had "a recent hemorrhage in the right eye and a small one in the left eye." Shortly thereafter she noticed that she was looking "through a veil" with her right eye. She was referred to a hospital, where after a short period of observation she was told that she had a "tumor of the brain" and was advised to undergo an "operation" (ventriculographic study). Alarmed at this information, she left the hospital against advice.

The past history was essentially irrelevant except for the radiation treatment of menorrhagia due to fibromyomas of the uterus one year previously. The patient was a housewife and had no occupational contact with heavy metals. She did not smoke. She drank no alcoholic beverages. Except for an occasional tablet of acetylsalicylic acid she took no drugs.

It was not until three days after admission that the following information was obtained from the patient, which she had suppressed previously because she did not think it relevant: Three days prior to the onset of the present illness she had noticed a few gray hairs in the front of her head and had gone to a

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From the private service of one of us (Keschner), the Montefiore Hospital for Chronic Diseases.

1. Stough, J. J.: Choking of the Optic Disks in Diseases Other Than Tumor of the Brain, Arch. Ophth. **8**:821-830 (Dec.) 1932.

beauty parlor to have her hair dyed a henna shade. The dye was applied without any preliminary cutaneous test. She remembered that her scalp "tingled and burned" after application of the dye and that some of the liquid trickled over her forehead, which became discolored. To remove the discoloration the beauty parlor attendant took some "ammonia" and vigorously removed the stain from this area. The irritation of the scalp disappeared after twenty-four hours, and the incident was dismissed from the patient's mind.

Examination.—The patient was well developed and obese, with a hypopituitary habitus. The hair was profuse and had a bright henna sheen, but there was no change in color at the roots that would lead one to suspect that it had been dyed. (The original color of the hair was brown.) The pupils were round, regular and equal and reacted normally to light and in accommodation. The right fundus oculi revealed papilledema of 2 D. The retinal arteries were attenuated, but there was no engorgement of the retinal veins. A few circumpapillary hemorrhagic spots were seen. The region of the macula was not involved. The left fundus showed a similar but less marked picture. The elevation of the disk on this side was about 1.5 D., and there were fewer hemorrhages. The blood pressure was 120 systolic and 80 diastolic. Otherwise the results of physical examination were negative.

Neurologic examination also gave negative results except for a slight facial asymmetry which the patient stated had been present all her life. On the Snellen charts central vision was found to be 20/50 in the right eye and 20/30 in the left. Perimetric examination disclosed a moderate generalized constriction of the visual fields, and stereocampimetric determination of the blindspots showed a normal blindspot on the left and an enlarged blindspot on the right, measuring from 0 to —10 degrees in its greatest vertical diameter and from 6 to 14 degrees in its horizontal diameter.

Laboratory Data.—The urine was normal. The value for hemoglobin was 90 per cent; the erythrocytes numbered 4,800,000 and the leukocytes 6,800 per cubic millimeter of blood; the differential count was normal. The value for blood sugar was 93 mg. per hundred cubic centimeters, and that for blood urea, 11.7 mg. Roentgenograms of the skull showed a slight increase in the digital markings of the vault but were otherwise normal. Roentgenograms of the nasal accessory sinuses were normal. The electroencephalogram showed normal and symmetric activity in all leads. Lumbar puncture yielded normal fluid under an initial pressure of 200 mm. of water (the patient was extremely apprehensive throughout the procedure). The Ayala index was 7.2.

Course.—The visual disturbances began to improve about one week after the onset and have continued to improve to date. When the patient was last examined, May 25, the hemorrhages in the left eye had entirely disappeared and those on the right were partly resorbed. The papilledema had receded bilaterally, and the disks were somewhat pale. The blindspots and the central vision were normal.

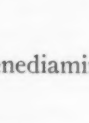
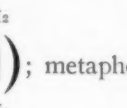
A sample of the hair dye was obtained from the beauty parlor at which it had been applied. The name of the product was "Glo-Rnz." It was described as a "dark brown, high color, tint rinse powder." The following caution notice appeared on the label, as required by the new federal Food, Drug and Cosmetic Act for all "coal tar hair dyes" [2]: "This product contains ingredients which may

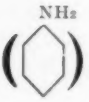
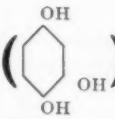
2. Federal Food, Drug and Cosmetic Act and General Regulations for Its Enforcement, Service and Regulatory Announcements, Food, Drug and Cosmetic, no. 1, Food and Drug Administration, United States Department of Agriculture, 1939, sect. 601A.

cause skin irritation on certain individuals and a preliminary test according to the accompanying directions should first be made. This product must not be used for dyeing the eyelashes or eyebrows, to do so may cause blindness."

COMMENT

The common dyeing agents in the coal tar hair dyes are all benzene ring derivatives. The substances most frequently employed are:

paraphenylenediamine  ; metaphenylenediamine  ;

aniline  , and pyrogallol  .

The toxic manifestations described for these substances are manifold and include almost every system in the body. The phenylenediamines are the most frequently used hair-dying agents for dark shades. They are also used in the fur-dyeing and feather-dyeing industries, for photographic developing, as textile dye intermediaries and in the vulcanization of rubber.[3] The dermatoses take first place in the order of frequency of toxic effects from these substances.[4] Asthma and gastrointestinal disorders are also described, as are cardiovascular failure and angioneurotic edema.[5] Sollmann[6] also listed vertigo, asthenia, diplopia, lacrimation, chemosis and exophthalmos. Close[7] reported a case of toxic labyrinthitis and subacute colitis traced to the use of a hair dye.

3. Beilstein, F. C.: Handbuch der organischen Chemie, Berlin, Deutsche Chemische Gesellschaft, 1930, vol. 13, p. 69. Hein de Balsac, F.; Agasse-Lafont, and Feil, A.: Aniline Poisoning in the Chemico-Pharmaceutical Industry: Cases, Progrès méd. **44**:935 (June 11) 1929.

4. (*a*) Levin, O. L.: Hair Dyes and Their Dangers, M. J. & Rec. **126**:204-206 (Aug. 17) 1927. (*b*) Brocq, L.: Dermatitis Venenata Due to Hair Dyes, Presse méd. **36**:1115-1116 (Sept. 1) 1928. (*c*) Spillman, L., and Weille, R.: Acute Dermatitis Bullosa wtih General Intoxication and Edema of the Glottis Due to Hair Dye with Paraphenylenediamine Base, Bull. Soc. franç. de dermat. et syph. **43**:214-218 (Feb.) 1936. (*d*) Hazen, H. H.: Paraphenylenediamine Dermatitis, Am. Med. **23**:296-299 (May) 1928. (*e*) Montrel, F. M.: Dermatosis Due to Ursol (Paraphenylenediamine), Venerol. i dermat. **7**:29-32 (Jan.) 1930.

5. Gonzales, T. A.; Vance, M., and Halpern, M.: Legal Medicine and Toxicology, New York, D. Appleton-Century Company, Inc., 1937, pp. 549-553.

6. Sollmann, T.: A Manual of Pharmacology, Philadelphia, W. B. Saunders Company, 1936.

7. Close, W. J.: A Case of Poisoning from Hair Dye (Paraphenylenediamine), M. J. Australia **1**:53-54 (Jan. 9) 1932.

Serious intoxications, both acute [8] and chronic,[9] have been reported. Three fatal cases [10] with autopsy reports are found in the literature. In all these there were acute, subacute or focal necrosis of the liver, hepatosplenomegaly, severe congestion of the viscera, anemia and toxic encephalitis.[10c]

Apparently the phenylenediamines and related aniline compounds exert their toxic effects either locally, as in the majority of the dermatoses, or on remote organs by direct absorption into the blood stream through the intact skin.[11] Regarding the distant toxic effects of these dyes following their absorption through the integument, MacKenna [12] stated: "It is most dangerous to attempt to remove the dye from the hair by the use of hydrogen peroxide or sodium thiosulphate as these measures may only intensify the symptoms." Israels and Sussman,[10b] in reporting a fatal case, described attempted removal of the dye from the hands with hydrogen peroxide as a possible aggravating factor. Baldridge [13] stated that the application of heat and ammonia during the giving of a "permanent wave" after the dyeing procedure may aid in the rapid absorption of the dye. This may also have been a factor in our case, in which a strong solution of ammonia was used to remove the dye from the forehead.

Numerous ocular disturbances ascribed to aniline dyes have been reported in the literature. Most of these effects have been local, due to topical application of the dye to the lashes or to the eyebrows. The

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8. Mollerström, J.: Acute Intoxication from the Use of Aniline Dyes, Acta med. Scandinav. **71**:73-81, 1929. Spillman and Weille.[4c]

9. Nott, H. W.: Systemic Poisoning by Hair Dye, Brit. M. J. **1**:421-422 (March 8) 1924.

10. (*a*) Unpublished case of chronic systemic poisoning from hair dye (paraphenylenediamine), from the medical service of Dr. L. Lichtwitz and the division of laboratories (autopsy no. 8393) of the Montefiore Hospital for Chronic Diseases. (*b*) Israels, M. C. G., and Sussman, W.: Systemic Poisoning by Phenylenediamine: Fatal Case with Pathological Report, Lancet **1**:508-510 (March 10) 1934. (*c*) Peters, H. R., and Sachs, M. S.: Systemic Poisoning Due to Synthetic Organic Hair Dye: Fatal Case with Autopsy, Ann. Int. Med. **12**:2032-2042 (June) 1939.

11. Gratioli, A. C., and Tenconi, J.: Poisoning by Cutaneous Absorption of Aniline: Case, Rev. sud-am. de endocrinol. **13**:809-811 (Nov.) 1930. Briedes, C.: Intoxication Due to Cutaneous Absorption of Aniline Dye, Semaine méd. **1**:104-105 (Jan. 4) 1934.

12. MacKenna, R. M. B.: Modern Cosmetic Preparations: Their Chemical Composition and Pathological Developments Attributable to Them, Brit. M. J. **1**:899-902 (May 17) 1930.

13. Baldridge, C. W.: Macrocytic Anemia with Aplastic Features Following Application of Synthetic Organic Hair Dye, Am. J. M. Sc. **189**:759 (June) 1935.

conditions described include blepharoconjunctivitis,[14] keratitis[15] and various less circumscribed types of ophthalmitis.[16] Toxic optic neuritis is a very unusual complication of aniline dye poisoning. Berger[17] reported a case of retrobulbar neuritis with a central scotoma for red and green and diffuse neurologic signs associated with aniline hair dye poisoning. Berger stated: "the presence of a peripheral toxic neuritis was indicated ophthalmoscopically in my case only by the appearance of hyperemia of the optic nerve." Veasey, cited by Berger,[17] reported a case in which "there were slight neuritic signs on the papilla" with bilateral central scotoma in an aniline dye worker. We have been unable to find a report in the literature of intraocular optic neuritis producing the picture of choked disks on this basis.

The mechanism of the production of this picture in our patient was probably direct absorption of the dye through the skin, with a generalized intoxication which was clinically most evident in the ocular fundus. How much of a role is played by a specific idiosyncrasy to the dye and a special vulnerability of the optic nerve in this case is a matter for speculation. It is possible that the attempted removal of the dye from the forehead with ammonia played an auxiliary role in hastening its absorption.

SUMMARY

A case of bilateral intraocular optic neuritis giving the appearance of papilledema is described. An erroneous diagnosis of tumor of the brain was made, which was discarded after a history of the application of a coal tar hair dye three days prior to the onset of symptoms had been obtained. Suggestions from the literature concerning the possible mechanisms of toxic absorption of the aniline dyes are reviewed.

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14. Harner, C. E.: Dermato-Ophthalmitis Due to Eyelash Dye, J. A. M. A. **101**:1558-1559 (Nov. 11) 1933. Greenbaum, S. S.: Dermatoconjunctivitis Due to Lash Lure, ibid. **101**:363 (July 29) 1933. Jamieson, R. C.: Eyelash Dye (Lash-Lure) Dermatitis with Conjunctivitis, ibid. **101**:1560 (Nov. 11) 1933.

15. McColly, A. W.; Farmer, C., and Loomis, E. C.: Corneal Ulceration Following the Use of "Lash Lure," J. A. M. A. **101**:1560-1561 (Nov. 11) 1933. Brav, A.: Toxic Ocular Manifestations from the Use of Eyelash Dye, Am. J. Ophth. **19**:894-895 (Oct.) 1936.

16. Bourbon, O. P.: Severe Eye Symptoms Due to Dyeing Eyelashes, J. A. M. A. **101**:1559-1560 (Nov. 11) 1933. Moran, C. T.: Bilateral Necrosis of the Cornea Following the Use of Hair Dye on Eyebrows and Lashes, ibid. **102**:286-287 (Jan. 27) 1934.

17. Berger, A.: Visual Disturbances Due to the Use of Hair Dye Containing Aniline, Arch. Ophth. **38**:397, 1909.

PENETRATION OF SULFANILAMIDE AND ITS DERIVATIVES INTO AQUEOUS HUMOR OF EYE

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The increasing importance of sulfanilamide and its derivatives as part of the therapeutic armamentarium of the ophthalmologist has made it imperative that more be known of the ability of these drugs to penetrate into the eye. This is of particular importance when they are employed in combating intraocular infections. These drugs exert their maximum therapeutic effect only with satisfactory concentrations at the site of the infection. Were this concentration impossible, employment of the drugs would be of little value.

Three drugs have been extensively used clinically in the treatment of infections, namely, sulfanilamide, sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). The literature on their use is too well known and extensive to warrant review of it here. Suffice it to say that their worth has been well proved.

Comprehensive studies on the concentration of these commonly used drugs in the aqueous have not as yet been done. Rambo [1] and Bellows and Chinn [2] showed experimentally that in rabbits and dogs sulfanilamide readily enters the aqueous. Mengel [3] and Luo and P'An [4] obtained similar results in human beings with abnormal eyes. Meyer, Bloch and Chamberlain [5] carried out studies on rabbits, using sulfapyridine. They found that this drug also freely entered the aqueous.

From the Department of Ophthalmology, University of Pennsylvania School of Medicine.

1. Rambo, V. C.: Effects of Sulfanilamide as Determined in the Eyes of Rabbits, Am. J. Ophth. **21**:739 (July) 1938.

2. Bellows, J. G., and Chinn, H.: The Distribution of Sulfanilamide in the Eye, J. A. M. A. **112**:2023 (May 20) 1939.

3. Mengel, W. G.: Determination of Sulfanilamide in Aqueous and Vitreous After Conjunctival and Oral Administration, Arch. Ophth. **22**:406 (Sept.) 1939.

4. Luo, F. H., and P'An, S. Y.: The Concentration of Sulfanilamide in the Aqueous Humor of Human Eyes, Chinese M. J. **58**:167 (Aug.) 1940.

5. Meyer, K.; Bloch, H. S., and Chamberlain, W. P., Jr.: Distribution of Sulfapyridine Between Blood, Aqueous Humor and Cornea, Am. J. Ophth. **24**:60 (Jan.) 1941.

Sadusk, Blake and Seymour,[6] as well as Spink and Hansen,[7] have shown that sulfathiazole enters the spinal fluid in only small amounts, while appearing in good concentration in other body fluids. For this reason its use is not advised for meningitis. It occurred to us that a similar low concentration might exist in the aqueous humor; so it was decided to compare the blood and aqueous levels of sulfanilamide and its commonly used derivatives, including a new one, sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine; sulfapyrimidine). This new chemical can as yet be obtained only for experimental use.[8] It was first synthesized by Roblin, Williams, Winnek and English.[9] This compound is said to be effective against experimental infections in mice due to staphylococci, streptococci, pneumococci and Friedländer bacilli, group B, as reported by Feinstone and others.[10] Reinhold, Flippin, Schwartz and Domm [11] reported that it behaves much like sulfapyridine in man; it is readily absorbed from the gastrointestinal tract and is readily diffused but is excreted slowly. The concentration in the spinal fluid averages 50 per cent of that in the blood. Flippin, Rose, Schwartz and Domm [12] expressed the opinion that it is of real value in the clinical treatment of pneumonia.

METHOD OF STUDY

Cats anesthetized with sodium amytal, 50 mg. per kilogram of body weight given intraperitoneally, were used throughout the experiments. Each was administered the sulfanilamide compound in one dose of 200 mg. per kilogram of body weight by stomach tube. Specimens of blood were taken by cardiac puncture six and twenty-four hours after the drug was given. Samples of the aqueous were taken by limbal puncture with a sharp, fine needle. Primary aqueous was taken

6. Sadusk, J. F., Jr.; Blake, F. G., and Seymour, A.: Observation on the Absorption, Excretion, Diffusion and Acetylation of Sulfathiazole in Man, Yale J. Biol. & Med. **12**:681 (July) 1940.

7. Spink, W. W., and Hansen, A. E.: Sulfathiazole, J. A. M. A. **115**:840 (Sept. 7) 1940.

8. Dr. Harrison F. Flippin supplied the sulfadiazine used in these experiments.

9. Roblin, R. O., Jr.; Williams, J. H.; Winnek, P. S., and English, J. P.: Chemotherapy: Some Sulfanilamido Heterocycles, J. Am. Chem. Soc. **62**:2002 (Aug.) 1940.

10. Feinstone, W. H., and others, cited by Plummer, N., and Ensworth, H. K.: Absorption and Excretion of Sulfadiazine, Proc. Soc. Exper. Biol. & Med. **45**:734 (Nov.) 1940.

11. Reinhold, J. G.; Flippin, H. F.; Schwartz, L., and Domm, A. H.: The Absorption, Distribution, and Excretion of 2-Sulfanilamido Pyrimidine (Sulfapyrimidine, Sulfadiazene) in Man, Am. J. M. Sc. **201**:106 (Jan.) 1941.

12. Flippin, H. F.; Rose, S. B.; Schwartz, L., and Domm, A. H.: Sulfadiazine and Sulfathiazole in the Treatment of Pneumonia, Am. J. M. Sc. **201**: 585 (April) 1941.

from the right eye six hours after the drug was given, simultaneously with the drawing of the first blood sample, and from the left at twenty-four hours, corresponding to the taking of the second blood sample. In some of the animals plasmoid or secondary aqueous was taken from the right eye thirty minutes after the primary aqueous was taken, or practically as soon as possible after its formation, while in some animals the secondary aqueous was not taken until the twenty-four hour interval had elapsed. We wished to know what concentration the drugs in question might reach in the secondary aqueous as compared with the primary aqueous and what might happen to it during the next few hours. The samples of blood and aqueous were analyzed quantitatively for sulfanilamide and its derivatives [13] by the method of Bratton and Marshall.[14]

EXPERIMENTS

1. Experiments were performed on 5 animals for verification of the work of Rambo [1] and Bellows and Chinn.[2] The drug used was sulfanilamide in an aqueous

TABLE 1.—*Diffusion of Sulfanilamide into Primary and Secondary Aqueous*

	Milligrams of Sulfanilamide per Hundred Cubic Centimeters					
	Six Hours After Ingestion of Drug			Twenty-Four Hours After Ingestion of Drug		
Cat	Blood	Primary Aqueous (Right)	Secondary Aqueous (Right), 30 Min. Later	Blood	Primary Aqueous (Left)	Secondary Aqueous (Right)
1	15.7	11.1	12.8	25.0	18.7	...
2	13.0	9.6	....	6.8	6.7	6.9
3	20.0	15.0	16.3	17.6	14.2	...
4	14.5	9.0	10.7	13.5	10.5	...
5	10.0	8.5	8.7	5.1	3.4	...

suspension, since this is readily absorbed from the gastrointestinal tract. Good blood levels (table 1) were obtained in six hours and were maintained for twenty-four hours, possibly because of the low output of urine, since no fluids were given during this period. The drug was found to enter the primary aqueous promptly and freely, as evidenced by the fact that similar levels were noted at six and twenty-four hours. An arithmetical mean of the values for the 5 animals showed the aqueous content to be 72.3 per cent of the blood content in six hours and 78.7 per cent in twenty-four hours (chart). The sulfanilamide content in the secondary aqueous reached 80 per cent of that in the blood.

2. Determinations of sulfapyridine were done in a similar manner on 5 cats, which confirmed the work of Meyer, Bloch and Chamberlain.[5] For these experiments the sodium salt of sulfapyridine was administered by stomach tube to most

13. These determinations were made by Helen M. Lynch, M.A., through cooperation with the Laboratory of Surgical Bacteriology of the Harrison Department of Research Surgery.

14. Bratton, H. G., and Marshall, E. K., Jr.: New Coupling Component for Sulfanilamide Determination, J. Biol. Chem. **128**:537 (May) 1939.

of the animals. According to the observations of Barlow and Climenko,[15] this preparation is very soluble and as a consequence is better absorbed than a suspension of the drug. The results (table 2) were comparable to those of the experiments with sulfanilamide, with relatively free penetration into the aqueous. The arithmetical mean value for sulfapyridine in the primary aqueous was 62.8 per cent of the blood value six hours after ingestion and 81.4 per cent twenty-four hours afterward. The sulfapyridine content of the secondary aqueous was 88.6 per cent that of the blood.

TABLE 2.—*Diffusion of Sulfapyridine into Primary and Secondary Aqueous*

	Milligrams of Sulfapyridine per Hundred Cubic Centimeters					
	Six Hours After Ingestion of Drug			Twenty-Four Hours After Ingestion of Drug		
Cat	Blood	Primary Aqueous (Right)	Secondary Aqueous (Right), 30 Min. Later	Blood	Primary Aqueous (Left)	Secondary Aqueous (Right)
1	12.6	8.3	...	10.3	9.3	8.6
2	9.8	6.5	...	8.3	6.5	6.8
3	6.1	4.1	5.1	11.2	8.4	...
4	11.5	6.5	10.6	15.0	12.3	...
5*	5.4	3.4	...	7.0	5.7	...

* Received an aqueous suspension of sulfapyridine.

TABLE 3.—*Diffusion of Sulfathiazole into Primary and Secondary Aqueous*

	Milligrams of Sulfathiazole per Hundred Cubic Centimeters					
	Six Hours After Ingestion of Drug			Twenty-Four Hours After Ingestion of Drug		
Cat	Blood	Primary Aqueous (Right)	Secondary Aqueous (Right), 30 Min. Later	Blood	Primary Aqueous (Left)	Secondary Aqueous (Right)
1	35.2	6.4	21.4	8.0	1.2	...
2	8.4	1.6	...	0.7	None	None
3	12.5	1.3	...	8.3	1.1	1.5
4	17.6	2.1	10.0	6.1	0.8	...
5	10.0	1.6	6.0	7.5	1.2	...
6	15.0	1.8	8.5	8.8	1.4	...
7*	1.3	None	0.6	3.6	0.7	...
8*	2.5	None	0.9	10.0	1.4	...
9*	7.9	1.8	5.0	10.0	1.8	...
10*	1.6	None	Faint trace	1.0	Faint trace	...

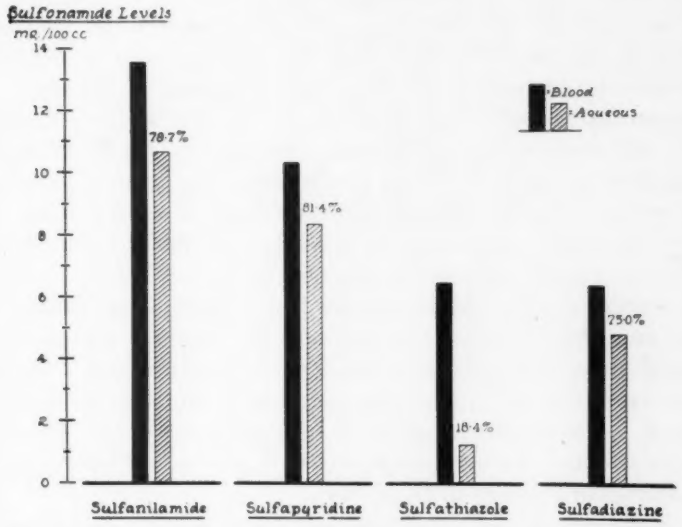
* Received an aqueous suspension of sulfathiazone.

3. Sulfathiazole experiments were carried out on 10 animals, since these data had not previously been reported. The sodium salt was used for all except 4 animals, for the reasons stated previously. Excellent blood levels (table 3) were obtained in the animals which received sodium sulfathiazole. Of extreme interest were the low values for sulfathiazole in the aqueous as compared with those in the blood. A comparison of the concentration of sulfathiazole in the aqueous

15. Barlow, O. W., and Climenko, D. R.: Studies on the Pharmacology of Sulfapyridine and Sulfathiazole Absorption, J. A. M. A. **116**:282 (Jan. 25) 1941.

with that in the blood, again from arithmetical mean values, showed that in the aqueous to be only 17.1 per cent of that in the blood at six hours and 18.4 per cent at twenty-four hours (chart). It was even held back somewhat from the secondary aqueous, since the level rose to only 59.2 per cent of the blood level. In the eyes in which the secondary aqueous was not taken until eighteen hours after the removal of an aqueous specimen, the level was of the same order as the primary level taken from the opposite eye at the same time.

4. Sulfadiazine studies were done on 10 animals. Aqueous suspensions of this drug were employed. It was found to diffuse significantly into the aqueous (table 4), but a definite lag in the aqueous level, as compared with a simultaneous blood level, was observed. Thus, at six hours the mean aqueous concentration



The levels of sulfanilamide and three derivatives in the blood and aqueous humor twenty-four hours after ingestion of single doses of the drugs. The figures indicate the relation of the aqueous levels to the blood levels (in per cent).

TABLE 4.—*Diffusion of Sulfadiazine into Primary and Secondary Aqueous*

	Milligrams of Sulfadiazine per Hundred Cubic Centimeters					
	Six Hours After Ingestion of Drug			Twenty-Four Hours After Ingestion of Drug		
Cat	Blood	Primary Aqueous (Right)	Secondary Aqueous (Right), 30 Min. Later	Blood	Primary Aqueous (Left)	Secondary Aqueous (Right)
1	4.8	1.6	3.2	7.6	5.0	...
2	5.6	2.9	4.2	4.8	2.4	...
3	2.7	1.7	...	5.2	3.0	3.4
4	0.7	Very faint trace	Faint trace	11.5	8.9	...
5	2.6	1.2	1.8	3.3	2.1	...
6	2.9	1.1	...	8.4	7.0	8.0
7	8.6	3.7	7.6	6.4	6.0	...
8	2.0	1.2	...	5.9	5.1	5.0
9	1.9	1.0	1.8	2.6	2.0	...
10	8.0	4.5	7.0	8.6	7.0	...

was found to be 48.8 per cent of the blood concentration, whereas the twenty-four hour aqueous level attained 75 per cent of the blood level. The level of sulfadiazine in the secondary aqueous was 80.3 per cent that of the blood.

COMMENT

The results obtained from the oral administration of sulfanilamide and sulfapyridine in cats confirm reports that these drugs enter the eye freely and can be expected to reach an effective concentration to combat infection.

On the other hand, strikingly low values of sulfathiazole were found in primary aqueous in relation to those in the blood. The quantities were so small that it is questionable that an effective therapeutic level could be attained. Furthermore, the ratio between the amount of sulfathiazole in the blood and that in the aqueous did not change significantly up to twenty-four hours after ingestion of the drug. For this reason little benefit could be expected from the systemic use of this drug in the treatment of intraocular infections. Also of interest was the fact that sulfathiazole, even in the secondary aqueous, penetrated poorly when compared with the other sulfanilamide compounds.

We observed some clinical support for the ineffectiveness of sulfathiazole against intraocular infection in 3 cases. One patient had a ring abscess of the cornea; another had a penetrating wound of the cornea, and the third had a postoperative cataract infection. The courses of these infections were unaltered by sulfathiazole. Since this series is not large enough to offer convincing evidence, more space is not taken for a detailed description of these cases, but the fact that sulfathiazole enters the eye very poorly is offered as a likely explanation for its therapeutic failure in these cases.

To make this report more comprehensive, studies were done with sulfadiazine, the most promising of the new sulfanilamide derivatives. It was found to enter the aqueous freely. Surely, if it fulfils present expectations, it can be used in the treatment of any intraocular condition for which chemotherapy is employed, since satisfactory aqueous levels should be expected.

SUMMARY AND CONCLUSIONS

The absorption of sulfanilamide and three derivatives and their penetration into the aqueous humor of the eye were studied in cats.

Sulfanilamide and sulfapyridine readily penetrated into the aqueous humor.

Sulfathiazole attained very small concentrations in the aqueous humor, as compared with its concentrations in the blood. This fact was offered as a contraindication to the use of sulfathiazole in the treatment of intraocular infections.

A new sulfanilamide derivative, sulfadiazine (2-[paraaminobenzene-sulfonamido]-pyrimidine; sulfapyrimidine), was found to diffuse into the aqueous humor in concentrations comparable to those observed with sulfanilamide and sulfapyridine.

NOTE.—During the preparation of this paper Bellows and Chinn [16] reported similar results on the penetration of sulfathiazole into the aqueous of dogs' eyes.

16. Bellows, J. G., and Chinn, H.: Penetration of Sulfathiazole in the Eye, Arch. Ophth. **25**:294 (Feb.) 1941.

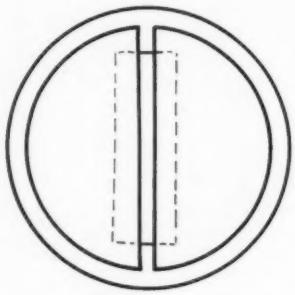
Clinical Notes

A METHOD OF PROCURING A NARROW BEAM OF LIGHT FOR OPHTHALMOSCOPY

BENJAMIN FRIEDMAN, M.D., NEW YORK

A narrow beam of light thrown on the fundus is of occasional value in ophthalmoscopy. Such a beam may be obtained by placing over the condensing lens of the ophthalmoscope a metal cap with a perforation in the form of a slit. The caps ordinarily available, however, cast too broad a band of light, because it is technically very difficult to cut a fine slit in the metal with production equipment.

I have been able to narrow the light streak by the use of two half-circles of thin copper foil, which I glued over the top of the metal cap.



Top view of the metal cap, showing the narrow slit formed by the superimposed semicircles of copper foil. The dotted line indicates the original slit in the cap.

One copper semicircle was placed so that its straight edge projected over the slit in the diaphragm; then the straight edge of the other semicircle was approximated as closely as necessary to form a narrow slit superimposed over the original diaphragm opening. Retroillumination is of help in gaging the width of the new slit. The straight edges of the semicircles must be perfectly true, and the evenness of their lines must not be disturbed by particles of glue.

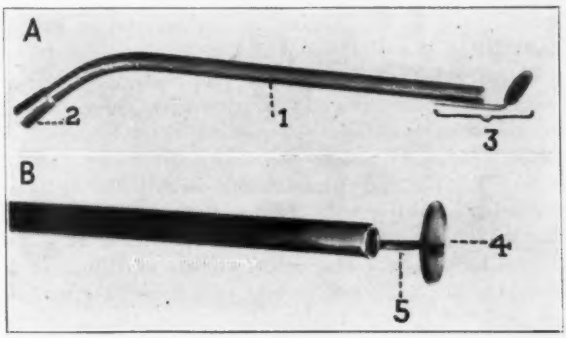
A COMBINATION CURET AND SUCTION SPOON FOR EVISCERATION OF THE EYEBALL

J. GOLDSMITH, M.D., NEW YORK

The instrument to be described was devised to simplify the operation for evisceration. Heretofore the removal of the diseased contents of an eyeball has been hampered by excessive bleeding from the choroid

and the ciliary body and also by the stringy, gelatinous consistency of the vitreous. The old method of curetting first and following this procedure by manual removal necessitated two separate steps. The device to be described acts in a dual capacity, so to speak, in that it provides a medium for directing the suction where it is needed after the diseased parts have been curetted. This procedure reduces the time element to a minimum. Several operations already have been completed in less than two minutes after excision of the cornea. To insure continued suction during the operation the assistant instills warm saline solution into the scleral cup.

The salient features of the device are shown in the accompanying photograph. A suction tube (*1*) made of brass with chromium plating is employed. It is 13 cm. long and makes a 145 degree bend 10 cm. from the intake opening, which has a 4 mm. bore. The walls of the tube at 10 cm. diverge to the out-take opening, which has a 5 mm. bore. This end (*2*) therefore is able to receive and hold more securely the flexible rubber tubing, which in turn is connected to a suction apparatus.



A, curet–suction spoon for evisceration. *B,* front view of the head of the instrument. (Constructed by W. H. Belz, of Belz, Opticians, New York.)

The head (*3*) consists of a concavoconvex circular disk (*4*) 11 mm. in diameter and 5 mm. thick. Its edge was purposely constructed with a fair degree of sharpness for the desired curetting effects. A thin metal bar (*5*) connects the disk, which is 6 mm. away, to the lower end of the main suction tube (*1*) and also forms an angle of 125 degrees with it.

LUDWIG'S ANGINA COMPLICATED BY ORBITAL CELLULITIS

STIRLING S. MCNAIR, M.D., JACKSON, MISS.

Orbital cellulitis and Ludwig's angina are two conditions concerning which there is little unanimity of opinion. This is true of the first condition because most authorities do not divide it into its clinical components, and it is true of the second condition because of loose

terminology. The term orbital cellulitis includes all inflammatory conditions of the orbit, from edema to phlegmon. I believe that ophthalmologists would have a better working knowledge of inflammations of the orbit if they divided all orbital cellulitic conditions as Ehlers[1] does, into (1) collateral orbital edema, which means an edematous condition of the contents of the orbit due to an inflammatory focus outside the orbit which has not yet advanced into the orbit; (2) subperiosteal abscess and preseptal orbital abscess and (3) phlegmon of the orbit. This classification of inflammatory conditions of the orbit also helps to determine the type of treatment to be instituted. Collateral orbital edema usually subsides without incision and drainage. In the presence of subperiosteal orbital abscess or of preseptal orbital abscess the orbit proper should not be opened, as the infection is outside and if the orbit proper is opened there is great danger of carrying infection into the orbit. The condition in the case that I shall report falls under the classification of collateral orbital edema.

REPORT OF A CASE

A white woman aged 49 was admitted to the hospital on Feb. 28, 1940. She had had a lower left first molar removed four days previously. On admission she was very ill. The left side of her jaw and neck down to the clavicle was swollen and very hard. The tongue was protruding from the oral cavity, and the dorsum of the tongue touched the roof of the mouth, which necessitated introduction of an airway to relieve the severe dyspnea. The temperature on admission was 99.4 F. The surgeon in charge decided that incision and drainage should be instituted immediately. An incision through the skin and the platysma muscle from the midline to the angle of the jaw was made on a line 2 cm. below the mandible. An incision was also made slightly anterior and parallel to the sternocleidomastoid muscle. No pus pockets were found, but drainage tubes were inserted and a moderate amount of seropus drained during the next few days. Culture showed hemolytic streptococci. Sulfanilamide was given intravenously, 60 grains (3.6 Gm.) in 1,000 cc. of physiologic solution of sodium chloride twice daily for two days and then lesser amounts. The patient also received two blood transfusions of 500 cc. each. She showed no improvement during the first week. She was semiconscious, and all medication had to be given intravenously. I was asked to see her on March 5, which was one week after her admission to the hospital, because of proptosis of the left eye. The result of my ophthalmologic examination was as follows: The right eye appeared normal. The left eye was proptosed 4 mm. straight forward, with nearly complete ptosis. There was a moderate amount of edema of the skin of the upper lid, with slight swelling of the skin and soft tissues of the lower lid and over the malar bone. There was a moderate amount of chemosis, especially in the upper portion of the conjunctiva. A moderate amount of pain was elicited by pressing the globe backward, and a sensation of doughy substance was imparted to one's fingers. The motility of the left eye was markedly limited, especially the action of the superior and internal rectus muscles, but there was no true paralysis. The pupils were small and equal; they reacted to light and dilated well with 2 per cent cocaine hydrochloride solution. Examination of the fundus of the right eye revealed the media to be clear; the disk was oval 90 degrees, of good color, well defined and with a

1. Ehlers, H.: Clinical Investigations on the Inflammatory Orbital Affections, Acta ophth., 1937, supp. 12, pp. 1-123.

moderate cup. The blood vessels and the macula were normal. The fundus of the left eye showed the media clear and the disk slightly blurred, especially nasally and above, but with no elevation. The veins were dark and slightly overfull. The arteries and the macula were normal.

The patient continued to improve, and ophthalmologic examination on March 7 revealed the proptosis and ptosis to be less; the chemosis was subsiding, and there was less limitation of movement. The eyes were examined again on March 12, and the exophthalmos was much reduced; very little ptosis was present, and there was full movement of all the extraocular muscles.

The patient continued to improve and was ready to leave the hospital on March 14, when she was seized with a coughing attack. She died four hours later. Autopsy was not performed, but death was thought to have been due to pulmonary embolism.

SUMMARY

A case of Ludwig's angina complicated by orbital cellulitis is reported.

A classification of orbital cellulitis as outlined by Ehlers [1] is given.

Orbital cellulitis due to an infected lower tooth must be extremely rare, as I have been unable to find any similar case reported in the literature.

REPLACEMENT OF THE FIXATION FORCEPS IN OPERATION FOR CATARACT BY THREE RECTUS MUSCLE BRIDLE SUTURES

FRITZ BLOCH, M.D., NEW YORK

For the last three decades ophthalmic surgeons have devoted a considerable amount of thought to facilitating and safeguarding cataract extraction. To avoid the pressure of the lids on the opened eyeball, especially in patients who squeeze the lids together, Van Lint [1] advised a temporary paralysis of the orbicularis oculi muscle by injecting procaine hydrochloride among the branches of the seventh nerve on the temporal orbital margin. In 1929, O'Brien [2] went a step further by completely blocking the peripheral portion of the seventh nerve in front of the external auditory meatus. In 1915, Axenfeld [3] recommended canthotomy to enlarge the field of operation, and in later publications he emphasized again and again the value of this measure. Use of a bridle suture through the superior rectus muscle is an additional improvement and is a simplification of Angelucci's [4] suggestion to fixate the eyeball during the incision with a specially constructed

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From the service of Dr. E. B. Gresser, Department of Ophthalmology, the Beth Israel Hospital.

1. Van Lint: Ann. d'ocul. **151**:418, 1914.
2. O'Brien, C. S.: Akinesis During Cataract Operation, Arch. Ophth. **1**:447 (April) 1929.
3. Axenfeld, T.: Klin. Monatsbl. f. Augenh. **54**:97, 1915.
4. Angelucci, A.: Arch. di ottal. **5**:71, 1897; **32**:138, 1925.

fixation forceps in the scleral area. Blaskowics [5] and Elschnig [6] recommended use of the bridle suture through the superior rectus muscle as a great step in making the cataract extraction safer, especially for the neophyte.

In my experience the greatest difficulty for the beginning ophthalmic surgeon is the incision. Often his trouble starts with the insertion of the fixation forceps. The teeth of the forceps may grasp only conjunctiva and some loose episcleral tissue instead of the muscle tendon. Conjunctival laceration and bleeding will develop occasionally. The eyeball begins to rotate in a wheel-like fashion, and the surgeon is inclined to use counterpressure against the eyeball with his fixation forceps instead of lifting the eyeball outward. Occasionally this causes subluxation of the lens and early loss of vitreous. Another point which creates difficulties is entanglement of the knife with the fixation forceps by a surgeon whose movements are not yet perfectly coordinated. Devoting his whole attention to the knife and its movements through the anterior chamber, such a surgeon frequently forgets the forceps in his other hand until the tip of the knife is caught in it.

These problems can be resolved by completely discarding the fixation forceps. After induction of the usual local and retrobulbar anesthesia, the lids are immobilized by a Van Lint or an O'Brien block; the lid speculum is inserted, and the palpebral fissure is enlarged by canthotomy. The tendon of the superior rectus muscle is grasped with a sharp-toothed forceps, and a no. 1 silk suture is inserted through the tendon. The same is done with the external and internal rectus muscles. The surgeon holds the suture of the superior rectus muscle; the assistant takes the lateral sutures, holding them tightly outward and slightly downward. The eye is then firmly fixed, and the incision is made easier and hence safer. The usefulness of these sutures is furthered when enlargement of the corneal wound by scissors is necessary; a slight pull on the contralateral suture greatly facilitates the enlargement.

A third advantage is lowering of the intraocular tension, with the probability of a negative pressure being created in the eyeball. If vitreous appears in the wound it should be possible by tension on the sutures to create a negative pressure inside the eye, thereby counteracting the momentary tendency of the vitreous to prolapse.

I have had no occasion to use this measure, since loss of vitreous has not occurred in the series of cataract extractions in which this type of fixation has been used. The lowering of intraocular tension has been recorded in the case of an 11 year old boy with traumatic cataract and a tension of 32 mm. of mercury (Schiötz). Twelve minutes after the retrobulbar injection of procaine hydrochloride the tension remained constant at 26 mm; after tightening of all three bridle sutures the tension was lowered to 22 mm.

A study of the literature does not reveal the routine use of bridle sutures through the muscle tendons. In 1919, van der Hoeve [7] recommended the insertion of silk sutures through the conjunctiva and the

5. Blaskowics: Klin. Monatsbl. f. Augenh. **52**:504, 1914.

6. Elschnig, A.: Klin. Monatsbl. f. Augenh. **52**:202, 1914.

7. van der Hoeve, J.: Klin. Monatsbl. f. Augenh. **62**:791, 1919.

superficial scleral layers near all four rectus muscles in the extraction of luxated lenses, in order to avoid loss of vitreous. In 1922, Elschnig,[8] mentioning this suggestion, commented: "If one should try this procedure, I would advise for numerous reasons to put the sutures (analogous to the bridle suture of the superior rectus) through the insertion of the four rectus muscles."

I do not claim that the three bridle sutures are indispensable to a successful cataract extraction, but the beginner or the surgeon who performs but a limited number of extractions in the course of a year, especially in a hospital where trained assistance is not at his disposal, will feel safer if he uses them and will avoid many complications which might impair the results of his surgical endeavor.

SUMMARY

The routine use of bridle sutures through the lateral and superior rectus muscles in intracapsular and extracapsular operation for cataract has demonstrated it to be valuable for firmer fixation of the globe, minimal injury to friable conjunctival tissue, greater freedom in the use of the knife and the scissors in corneal sections and better control of intraocular tension and the vitreous body.

8. Elschnig, A.: Augennärzte Operationslehre, in Axenfeld, T., and Elschnig, A.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1922, vol. 2, p. 1210.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

SIMULATED BLINDNESS

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The sudden emergency which has resulted in a history-making peace time conscription in the United States stimulated the need of a review of tests for disclosing simulated blindness, which will undoubtedly arise among the draftees. Malingerers pretending blindness can be divided into two general groups: (*a*) those who are clever and study the available and well known tests prior to being examined and (*b*) those who desire to magnify a defect but are too naive to deceive a conscientious examiner for any length of time.

A thorough clinical study of the eyes, including a retinoscopic examination, must be conducted to prove the absence of a significant organic lesion. The patient should be given every consideration, should be listened to sympathetically and should not be allowed to entertain any idea that he is under suspicion. He may be overconfident and overanxious to cooperate. Leading questions are the order of the day. Tests should be repeated if necessary and on different days in order to confuse the examinee, who will not be able to remember in detail his responses on previous occasions. Trickery can be overcome by trickery; falsifiers convict themselves. Patience, sagacity, thorough knowledge of the applied tests and modifications of the old tests will suffice to expose most malingerers.

Simulated blindness of one eye can be demonstrated objectively and subjectively. The objective methods are first pursued. They consist of (*a*) close observation of direct and consensual pupillary reactions and (*b*) checking of the ocular movements in relation to the visual axis. I shall discuss these methods separately.

A proper investigation of the condition of the pupils must be preceded by an examination to determine the presence or absence of synechiae, miosis and response to sensory stimuli. After pathologic changes of the iris have been ruled out, the next procedure is to study the influence of light on the suspected eye. Place the patient in a dark room. Focus a beam of light on the healthy eye and note the degree and rapidity of contraction of the pupil. Screen this eye and flash the beam into the

other eye. If the latter eye has an unresponsive retina, the healthy eye will not react. If the suspected eye is amblyopic, the pupil of the other eye will react in proportion to the intensity of the light and the degree of amblyopia. Special attention must also be given to the shape, position and size of the pupils. An amaurotic or amblyopic eye may well have a dilated pupil. The examiner must bear in mind the possibility of mydriasis induced by the aid of a drug. Under such circumstances direct and indirect (consensual) stimulation, of course, show a fixed pupil in the suspected eye, while the healthy eye may react. A point of importance is the fact that if the suspected pupil remains inactive when the eye is exposed to light while it contracts under the influence of convergence-accommodation, it indicates with reasonable assurance the existence of unilateral amaurosis. If the eye is amaurotic or decidedly amblyopic, it may well deviate when the vision of the fellow eye is directed toward a fixed object. If there is no manifest divergence, a simple screening test at about 3 meters will uncover any latent tendency.

The second of the objective methods, the direction of the visual axis, can be illustrated by the von Welz test.[1] This procedure consists in the placing of a 10 to 20 degree prism, base out, before the suspected eye and asking the patient to read. If the patient has appreciable vision in this eye, the eye will deviate inward and removal of the prism will result in motion outward to maintain binocular single vision. This is all quite beyond the control of the subject. The test presupposes binocular single vision.

Smith and Jackson utilize the same test but employ a 6 degree prism, base out, and have the patient fix on a candle or a point of light at 6 meters.[1] The subconscious desire for binocular single vision will bring about the inward rotation of the suspected eye and its return to primary position on removal of the prism.

Duane suggested [2] that the patient be asked to read aloud rapidly; suddenly a 4 degree prism is placed, base down, before the suspected eye. If the eye is amaurotic or very amblyopic there will be no effect. Otherwise confusion will result and the reading will be poor or impossible.

Bach [3] described an objective method called the optokinetic nystagmus test. This will unmask both simulated and hysterical blindness. Optokinetic nystagmus is that type which occurs with nystagmus-like twitchings or movements of the eye when the subject looks at such

1. Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1900, vol. 4, p. 861.

2. Fuchs, E.: Text Book of Ophthalmology, translated by A. Duane, ed. 8, Philadelphia, J. B. Lippincott Company, 1924, p. 241.

3. Bach, K.: Testing of Optokinetic Nystagmus for Detection of Simulated Blindness, Klin. Wchnschr. **14**:1505, 1935.

objects as those seen from a moving train or car. The instrument used is the optokinetoscope, which can be easily constructed as follows: A vertical drum, about 25 cm. in height and 19 cm. in diameter, has alternate black and white vertical stripes, each 25 mm. wide, painted on it. The drum is mounted on a central rod so that it may be slowly revolved in the vertical position. A suitable handle is attached to the central rod below the drum, and this is used to turn the drum. Slowly turning the drum before the patient's eyes will induce nystagmus if the eyes have vision. The nystagmus results from the fact that during movements of objects or of the observer the objects are seen only when the eye is fixed momentarily on one object after another. In the performance of the test, cover the good eye and have the patient face the instrument at the usual reading distance. Slowly rotate the drum after instructing the patient to try to see whether it moves or not. A seeing eye will find it almost impossible to avoid looking at the drum, and nystagmus will result. This positive result proves the fraud. Evans suggests that the examiner watch the good eye with the ophthalmoscope while the patient faces the revolving drum. Any nystagmus will be quickly observed in this manner.

The subjective methods of detecting simulated blindness are numerous and depend on the principles of optics and of physiology. The tests are all dependent on the use of plano and concave mirrors, prisms, screens, visual acuity charts, etc. The more ingenious the examiner, the more tricky and complicated the tests have become. As Baudry stated, "The eye is, par excellence, the organ of illusions, and the methods of deceiving it are numerous." [1] (Any magician will happily confirm this.) It is most important, and this is a great weakness of nearly all the subjective tests, that the patient should not be allowed to close either eye during the examination.

The Javal-Cuignet Method, or bar test,[1] consists in holding 35 mm. from the eyes a sheet of paper on which are dots, figures and printed letters. A pencil or a finger is held midway in the median line between the eyes and the paper. If the subject reads the entire line he must see equally with both eyes; otherwise a portion of the field of vision would be missing. With a graduated scale of letters and figures the approximate visual acuity may be determined. In the proper application of this test the head and the printed test sheet must be held immobile. The interposed pencil or finger may be moved to confuse the subject further. Martin modified this test by using a box with two eye pieces in front and a movable bar 1 cm. wide in the center, with the test type on the back wall. Barthelemy further complicated it by using prisms and mirrors. Driver simplified the test by using a vertical ruler 4 cm. wide and two Snellen test types. The ruler is held so that it acts as a screen that hides the right test type from the left eye and the left test type

from the right eye. If the patient reads both scales his dishonesty is exposed. The examiner must be sure that both eyes are kept open while the tests are being performed.

Certain tests [1] in which mirrors are the essential features strive to defeat the patient by having him see with the so-called amblyopic eye what he believes he is seeing with his good eye. These tests involve the construction of an apparatus sometimes called a pseudoscope. Fles devised the first one in 1860, and the modifications have been numerous. Few of these tests indicate the actual visual acuity of the suspected eye. Concerning the pseudoscope, it is only fair to mention one such instrument, devised by Chauvel, with which the visual acuity can be determined by the use of printed letters on a sheet of glass which acts as the posterior wall of a rectangular box. The examiner is able to watch the patient's eyes and can cross and uncross the images at will. For details of construction of the pseudoscope the reader is referred to the article by Baudry (in Norris and Oliver [1]).

A further group of subjective tests has been elaborated by the employment of uncolored lenses. A complete objective refraction and study of the suspected eye must be accomplished first. The proper correcting lenses should be placed before the patient's eyes if he is not emmetropic. The simplest procedure (Harlan [4]) is to add a + 10 to 20 D. lens before the good eye and a plano or a very weak minus sphere before the "defective" eye. The patient is then instructed to read. If he does so, he is obviously using the eye that is supposedly amaurotic or amblyopic. Another method, said to be useful, is to render the normal eye myopic with a + 6 sphere, placing a plano before the second eye. The patient is asked to read test type that is held close to his eyes. The type is gradually withdrawn from him until it is beyond the focus of the seeing eye, about 17 cm. Continued reading at this distance discloses the fraud, and the size of the print read will measure the visual acuity of the allegedly blind eye. A Lebensohn chart would be very valuable for this purpose; however, it may be well to remember that, as Duke-Elder stated, "Identical visual angles do not necessarily give the same visual acuity."

Baroffio and Baudry suggested the use of physostigmine, atropine or pilocarpine in the good eye and distilled water in the "defective" eye.[2] These drugs would obviously impair the vision of the normal eye, and if the patient could continue to read he would be using his other eye. The disadvantages and unreliability of this method are self evident.

Cylinders have also been put to work in an effort to detect malingerers. Among the methods used [5] are those of Kugel, Segal and

4. Harlan, G. C.: A Simple Test for Simulated Monocular Blindness, Tr. Am. Ophth. Soc. **3**:400, 1882.

5. Simulation of Blindness, in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913, vol. 2, p. 1193.

Jackson. Kugel places two strong cylinders respectively before each eye at right angles to one another and has the patient look at a candle flame in a darkened room. A luminous cross would be visible if both eyes could see. Maddox rods can be used for this test. Segal uses the same method, but with + 1 D. cylinders, and has the patient read. If reading is as easy with both eyes as with the normal eye alone when not fitted with glasses, it may be deduced that the patient is simulating. Jackson places the two cylinders at right angles to one another before the normal eye and covers the second eye with a plano or correcting lens. He directs the patient to read and rapidly rotates the cylinders, excluding the normal eye from useful vision. Jackson's method is superior to the others in that it will ascertain visual acuity.

Prisms have been and still are one of the best sources for subjective as well as objective tests to demonstrate simulated blindness. In producing diplopia or triple vision, the examiner must be careful to impress the patient with positive statements and not with vague questions. The patient must not be asked whether he sees two images; he should be asked: "Is one image directly above the other?" or "alongside the other?" etc. Von Graefe in 1867 devised a method of creating monocular diplopia by placing a strong prism base or edge (the edge is preferable because it is less noticeable) before the sound eye in such a manner as to bisect the pupil, the suspected eye being covered at the time. The prism is then quickly moved over the whole pupil while the second eye is simultaneously uncovered. Diplopia means binocular sight, and the malingerer is exposed. Galezowski utilized the double refractive lens of Arago to create alternate monocular and binocular double vision, while Fröhlich went a step further and by using a double refractive prism of 14 degrees produced monocular and binocular triple vision. However, these powerful prisms create differences in the images as seen by the two eyes and may readily aid the malingerer in his efforts at deception. Baudry developed a box arrangement containing a triangular prism with a special base. This instrument can be so manipulated that the patient cannot possibly know which portion of the prism is before his good eye; furthermore, a red instead of a white light is used in order to prevent dissimilar images for the two eyes.

With the aid of two prisms of 10 degrees each, a simulator may be easily upset as follows: Place one prism base down before the good eye and have the patient look at a light about 3 meters away; he will see two images. Place the other prism in the same way before the second eye and ask the patient whether he still sees double. If he says yes, he is malingering.

Todd[6] advocated the following procedure: A double prism with bases together is placed over the seeing eye in such a way as to produce

6. Todd, F. C.: Malingering (Pretended Blindness), Ophthalmology **9**:81, 1912.

monocular diplopia. If the patient sees three lights, he is seeing with the allegedly blind eye. If he denies seeing two lights when the prisms are properly placed, it is evidence of dishonesty. Prisms have been tried in other tests, even colored prisms. One supposedly effective method is to place a prism base up or base down before the good eye and direct the patient to walk up or down a staircase. If he has binocular vision he will be in great difficulty unless one eye is closed. The literature contains so many tests in which prisms are the important elements that it seems as though no new applications could be devised. One or two tests should be studied thoroughly by the examiner until he is very proficient; this is his best guarantee of success in the detection of the malingerer.

Stereoscopic tests have certain disadvantages. These methods of examination demand binocular single vision and not too great a degree of anisometropia. Further, it is difficult to detect the malingerer if he momentarily closes one eye. The tests depend on the use of cards that have the printed material so arranged that each eye sees certain portions exclusively. Obviously, many types of pictures and letters can be used, the tests varying accordingly. A practical method is that advocated by Todd. Place a Worth amblyoscope, previously arranged so that if normal eyes look through it crossed images will be seen, on a table where the examiner will be able to see that its tubes do not cross. Now put a picture into each tube, allowing the claimant to witness the procedure. If he does not close his eye he will not know of the crossing of the images and will state that he sees only the image on the side of the normal eye. Thus his deception will be disclosed. Snell [7] described certain slides that have combinations of letters, words or numbers so arranged that adjusting the tubes of the amblyoscope will cause many different possible results. Of course, this can be very confusing to the patient. Haseltine [8] has made use of this same principle and has stated the belief that it is more effective if letters of the alphabet are placed on the slides with red additions on certain of the letters. For instance, a black O has a red tail to make it a Q; a black F has a red bar to make it an E, etc. A dark smoked glass is placed before the blind eye and a red one over the normal eye. If the examinee reads the letters as modified by the red additions, he certainly is seeing with the allegedly blind eye. The optical principle that a monochromatic colored glass allows similarly colored rays to pass through it and stops rays of the complementary color has led to a group of tests dating back to 1870. Thus, if dull red and green letters on a dull black background are viewed

7. Snell, A. C.: A Treatise on Medicolegal Ophthalmology, St. Louis, C. V. Mosby Company, 1940, p. 231.

8. Haseltine, S. L.: Tests for Vision Malingering, Eye, Ear, Nose & Throat Monthly **17**:49, 1938.

through a red glass, the red letters will be visible while the green ones will be black and invisible. Snellen applied this fact in his test for malingering. He placed the red glass before the sound eye, and if the green letters were seen the patient was certainly a fake. The test can be reversed and a green glass used instead of the red, causing the red letters to be invisible to the sound eye. Visual acuity can be determined at the same time if graduated letters are used. Snellen constructed a board with test types which were alternately red and green. A pair of colored spectacles, with one lens red and the other green, is placed on the patient, and he is directed to read rapidly. If he is not blind (or too clever) or if the colors are not defective, he will read both red and green letters. If one eye is amaurotic or amblyopic he will read the red or the green letters accordingly. A modification of this test depends on the fact that, if the background is white the red letters will be invisible (the white background becomes as red as the red letters) and the green ones will be black when viewed through a red lens. Write letters on a white paper with red and black pencils alternately, but do not press marks into the paper. Direct the suspect to read the writing rapidly. A red glass is interposed before the sound eye, making the red letters invisible to this eye. If all the letters continue to be seen the patient is simulating (Haseltine [8]). Duane directs that the patient read aloud; as a further check on him, he suddenly places a prism of 2 D. base down before the poor eye. If this eye is amaurotic or amblyopic the examinee will not be troubled; however, if he sees fairly well with this eye the sudden creation of vertical diplopia will cause confusion, and he will involuntarily stumble and perhaps stop reading.

Before testing any male subjects with colors one must be certain to rule out color blindness, which is prevalent among men. All lenses and colors used should be of the finest quality obtainable.

Von Szily [9] makes use of the physiologic blindspots to trap a simulator. Three round spots about the size of a silver quarter are appropriately placed on a wall at the level of the eyes. If the central one is fixed at the appropriate distance all three are seen. If a central diaphragm is placed in the sagittal plane to act like the diaphragm of the stereoscope, the image of the right lateral spot will fall on the blindspot of the right eye and the image of the left lateral mark made on the wall will fall upon the blindspot of the left eye if the distance of the patient from the wall is appropriate. Under these circumstances a normal two-eyed person sees but one spot, the fixing spot. If the diaphragm is removed, again three spots are seen. If this test is repeated for a one-eyed person, three spots are visible until the proper distance is used, whereupon the

9. von Szily, A.: The Blind Spot in the Use of Detecting Unilateral Simulated Blindness, Klin. Monatsbl. f. Augenh. **65**:1, 1920.

image of the test spot on the side of the good eye falls on that blind spot and only the other two spots are seen. If the diaphragm is now placed, only the fixing spot is visible, as the spot on the blind side is excluded from the field of the good eye by the diaphragm and the spot on the side of the good eye casts its image on the blindspot. Removal of the screen immediately brings back the second spot (the one on the blind side), and if the patient is moved forward or back the third spot will appear.

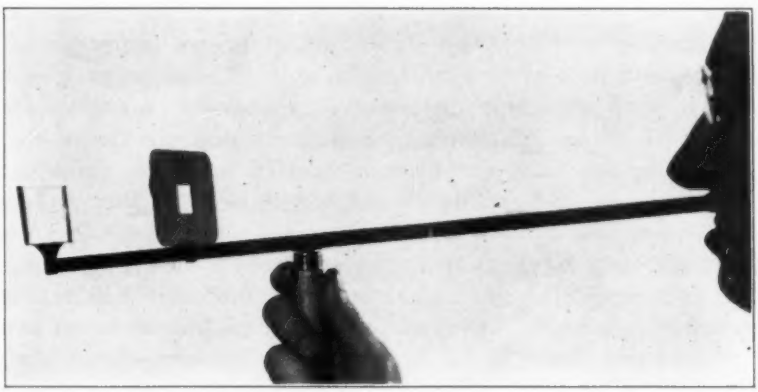
When a simulator is put through this test, he alleges that he sees two spots when the diaphragm is placed in position, because he thinks that the spot on the side of the blind eye should be invisible to the blind eye. By doing this both at the proper distance for the lateral images to fall on the blindspots and nearer to and further from the test surface, a satisfactory test is easily carried out. This test may also be carried out with most of the apparatus on which the blindspots are indicated: the Peter campimeter, the Duane screen and the stereocampimeter. The ordinary hand stereoscope may also be used. If monocular apparatus is used, a mark is made at the fixing point and also on each blindspot outline. The apparatus is placed with the fixing point opposite the bridge of the nose, and the distance should accord with the scale for which the blindspot locations are mapped. If the diaphragm is now placed the normal two-eyed person will see but one spot, the fixing spot, but the malingerer will assert that he sees two; a one-eyed person, but one. The stereoscopic method is somewhat different but has many interesting variations, which can be used to confuse the most astute. The diaphragms cannot be moved, but a single fixing mark can be made before the good eye and a blindspot mark before each eye. With this arrangement, the two-eyed person sees one mark, the fixing mark. A one-eyed person sees the same mark, on which he fixes. The malingerer says that he sees two marks. Instead of a fixing mark, a white test object can be placed at the fixing point of the good eye and the lateral spots can be made to appear and disappear by asking the patient to follow the fixing test object as it is moved a few degrees up or down or right or left. As soon as the relation between the fixing point and the blindspots is changed, the lateral images fall on the retina, and all three spots should be seen. None but those specially trained in this particular test can think fast enough to deceive the examiner. A personal trial will show the possibilities of the test when used in this way.

Lloyd [10] cautioned that there are a number of persons who do not have binocular distance vision and that there are others who do not use both eyes at the near point. Stereoscopic testing is really a test at optical infinity, but 1 meter of distance is certainly not a far test. This must

10. Lloyd, R.: Personal communication to the author.

be taken into consideration when using the von Szily test, because *positive* evidence that will prove the use of both eyes is obtained only when both eyes are so alined that the lateral images fall on the blindspots. If the patient has a deviating eye, an image is suppressed and the evidence becomes *negative;* it is known only that he is using one eye in the test, and it is not known whether the eye is blind or merely suppressing an image. The eye with the suppressed image may have very good vision, with or without correction when tested alone. (Differences of opinion exist whether suppression is characteristic of the macula or of the extramacular portion of the retina.)

To make the test accurate, the size and location of the lateral blindspot marks placed on the wall must have the same relation to the fixing spot that the normal blindspots bear to the common fixing point. Also,



Bishop-Harman diaphragm test.

to allow for variations in size and location of the blindspots and to make the test easy to apply for the varying degrees of intelligence encountered, the lateral spots on the wall should be made about one-half the size of the normal blindspots as projected on the test surface at the distance used.

The stereoscope may be utilized to detect simulated blindness. A series of stereograms must be used, beginning with simple types having similar characters on both sides; the image is the same to either eye. As the patient becomes familiar with the instrument, he is worked up to the more complicated types, and if he is not too clever he may soon reveal binocular vision.

The Bishop-Harman diaphragm apparatus (see illustration) is an excellent device to detect binocular vision and allows constant observation of the patient's eyes.[10] The images are crossed and are seen at the reading distance. A series of test cards with graduated letters is run through. The claimant, if deceived, readily believes that the images

on the right belong to the right eye and vice versa. In this way the visual acuity of the so-called blind eye may be quickly determined. Lloyd [10] suggested that to confuse the claimant further he may first be asked to use the amblyoscope, which has been so adjusted that the images are uncrossed. The patient has this fact firmly fixed in his mind and will not expect anything different from the Bishop-Harman instrument, which, of course, crosses the images. Evans has constructed a modified stereoscope that accomplishes the same purpose.

A new test with polaroid disks can be credited to Gradle.[11] It depends on the principle that light polarized in any given meridian by a polaroid screen is selectively absorbed by an analyzing screen whose axis is at an angle to the axis of the polarizing screen. Three disks of polaroid, 36 mm. in diameter and 2 mm. thick, are needed. They are held in the ordinary trial frame with the handle corresponding to the polarizing axis. One polaroid disk is placed before each eye with the polarizing axis horizontal. Before the good eye the third disk is placed with its axis also horizontal. The patient is then directed to read the smallest possible line on the Snellen chart with both eyes open. Immediately the third disk is rotated so that its axis is vertical for a short time. This makes the chart invisible to the good eye, and if reading is continued uninterrupted, the allegedly blind or amblyopic eye is proved to see and its visual acuity may be measured; correcting lenses can be used with the polarizing disks.

Bakker [12] described a device with which a Snellen chart can be illuminated with red, green or yellow rays from a sodium lamp. The patient is seated opposite the chart, wearing his correcting lenses if ametropia is present. The examiner pretends to concern himself only with the patient's good eye but warns him against closing one eye. The examiner then puts a green glass over the patient's good eye and turns the green light on the Snellen chart. The patient reads with his good eye. If he should close the "bad eye," he finds only that he is reading with his good eye. Then a red glass is put over the good eye and the red illumination turned on. Again the patient reads with his good eye. Another red glass is put in front of the good eye and the sodium lamp turned on. The rays emitted by this lamp are absorbed by the red glass in front of the patient's good eye. Thus he unknowingly reads with his "bad" eye.

The aforementioned tests can be used to detect simulated blindness of one eye. Many are of service in determining the visual acuity of so-called amblyopic eyes. Among the latter group are the Javal-Cuignet

11. Gradle, H. S.: Another Test for Malingering, Am. J. Ophth. **20**:300, 1937.

12. Bakker, A.: Sodium Light as an Aid in Detection of Malingering in Ophthalmology, Arch. f. Ophth. **139**:267, 1938.

or bar test, Chauvel's pseudoscope test, Harlan's test and the Bishop-Harman test. A most useful trick is to have the patient read an ordinary chart at 3 meters and then read the image of a similar chart (but with reversed letters) that has been placed above his head in a mirror at a distance of 3 meters. Obviously the distance becomes 6 meters, and if the claimant reads the same line as previously he is malingering.

By changing the size of the visual angle the fraud may be uncovered. The size of the visual angle increases as the distance between the patient and the test card decreases. Each time the card is placed at one-half the previous distance, the size of the visual angle is roughly doubled. If, for example, the distance is reduced from 20 to 10 feet (6 to 3 meters) the claimant should be able to read test letters one-half the size of those read at 20 feet. Snell [7] pointed out that if the visual acuity is 20/100 at 20 feet, it should be 10/50 at 10 feet and 5/25 at 5 feet: each test in this case should maintain a 1:5 ratio. Failure on the examinee's part to adhere to this principle will reveal his fraud. The test can be applied at the near reading distance, but it is not very satisfactory in this case, because the print must be held at exactly measured distances from the eye and also because the patient may not cooperate well owing to his familiarity with ordinary printing.

During the last war, Evans and his associates made use of the knowledge that many simulators believed that vision limited to the two top lines of the chart (20/200, 20/100) was disqualifying.[13] Some charts were prepared by simply cutting away these figures and then submitting the reduced charts to the examinees. Many of the latter blithely read the two top lines, which required 20/70 and 20/50 visual acuity, and thus exposed their duplicity.

The study of the central and peripheral fields will often reveal decided discrepancies in cases of fraudulent intent. The malingerer usually contracts his fields concentrically and tends to exaggerate this contraction far out of proportion to the actual acuity that he admits. Angioscotometric examination will give further corroboration of the accuracy of the peripheral and the ordinary campimetric fields. Repeated tests with different instruments and screens and on different days usually will so confuse the claimant that his attempted deception will be admitted when he is confronted with the recorded results. Patience and ordinary care on the part of the examiner are all that are required.

Simulated blindness of both eyes is a rare phenomenon, which, however, may be met in cases of hysteria. A careful history of the mode of onset and the duration of the "blindness" would be of great help to the examiner. The patient may wear smoked glasses or avoid bright lights, contrary to the behavior of the truly blind or of patients with

13. Evans, J. N.: Personal communication to the author.

serious bilateral amblyopia. Ask the patient to cross a room in which articles of furniture are placed as obstacles. He may avoid them by elaborate and exaggerated motions or walk directly into them forcefully. The simulator may sign his name too well or in a grossly irregular manner. He should be watched by constant attendants. Total blindness can be readily disproved by the objective prism test and the optokinetic test. Ophthalmoscopic and pupillary reflex studies usually give a good clue to perception of light in contradistinction to total blindness. If partial vision with both eyes is admitted, patient application of the various tests mentioned will probably trick the subject into disclosing his attempted fraud.

In 1918 the War Department encountered many men with simulated blindness who presented themselves to the draft board examiners. As a result of this wide experience, a set of specific tests has been elaborated and prescribed in Mobilization Regulations 1-9.[14] These regulations include the polaroid disk test as described. Three prism tests are mentioned: (1) a 6 D. prism, base down, is placed before the sound eye, admitted diplopia proving binocular vision; (2) a 10 D. prism, base out, is placed before the "blind" eye, which causes the eye to turn in if binocular vision exists, and (3) a 10 D. prism is used to produce monocular and then binocular diplopia as described. Also mentioned are the test with colored glasses and letters; the Harlan test; the stereoscope test; the bar test; the mirror test with special cards; a careful study of the refractive state of the eyes, and a consideration of the candidate's occupation.

It has been my aim to present a series of tests for simulated blindness the application of which would aid in uncovering any attempt to defraud or to establish a false record of defective vision. I should like to reiterate that the examiner should thoroughly familiarize himself with the tests he intends to apply, for some of the examinees may be more acquainted with the tests than the examiner is. Nothing can be well done if it is not well prepared.

14. Standards of Physical Examination During Mobilization, Mobilization Regulations 1-9, United States War Department, 1940.

Correspondence

POLARIZATION

To the Editor:—Apropos of Dr. David G. Cogan's article on "Some Ocular Phenomena Produced with Polarized Light," in the March 1941 issue of the ARCHIVES (ARCH. OPHTH. **25:** 391 [March] 1941), I wish to make a few remarks concerning polarized light.

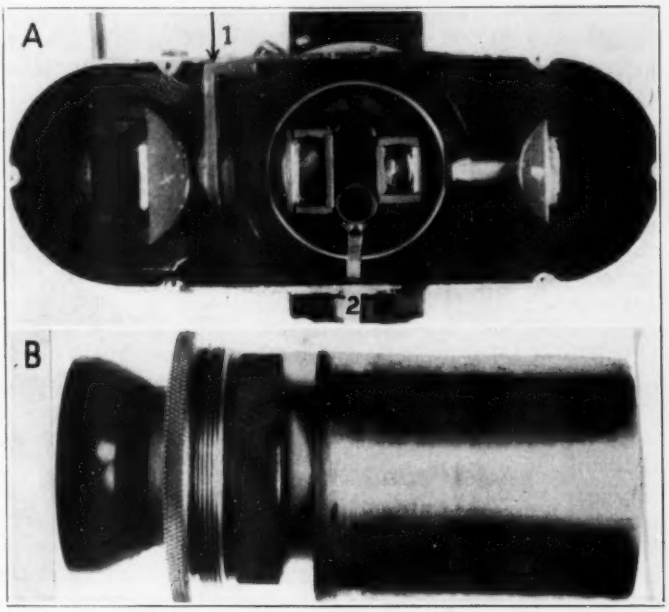
Prior to the revolutionary invention of polaroid by Edwin H. Land, of the Polaroid Corporation, investigators in the field of polarized light employed Nicol prisms of calcite. Polarizing accessories were constructed for the biomicroscope and the ophthalmoscope, and several papers have appeared giving details of ophthalmic studies. In fact, a Nicol polarized ophthalmoscope devised by Cardell in England has been sold commercially. Nearly all the problems of the optics of polarized light had been worked out prior to Land's invention—which provided the long-sought wide aperture polarizing contrivance—and now a resurgence of interest in this absorbing field of optics is well on the way.

Professor B. Strampelli, of the University of Rome, wrote a monumental work in 1935 (*Boll. d'ocul.* **14:** 34, 1935) concerning his researches in the biomicroscopic and histologic examination of cataract with polarized light. He was familiar with the lenticular interference cross and the various chromatic birefringent effects which were obtainable.

Shortly after the publication of his work on the lens Strampelli devised a polarizing ophthalmoscope embodying Nicol prisms. In his paper (*Boll d'ocul.* **14:** 595, 1935) he described the appearance of the anterior segment of the eye as seen with crossed polarized light and with plane polarized light. He presented good colored illustrations, which apparently anticipated Dr. Cogan's. Furthermore, Lemoine and Valois (Bailliart, P.; Coutela, C.; Redslob, E., and Velter, E.: Traité d'ophthalmologie, Paris, Masson & Cie, 1939, vol. 2, p. 885) gave a résumé of Strampelli's paper.

The production of the interference figure depends primarily on the use of convergent (or divergent) polarized light. Crystallographers explain the interference cross somewhat differently than did Dr. Cogan (Wood, R. W.: Physical Optics, New York, The Macmillan Company, 1929, pp. 321-322). Assuming that the corneal phenomena may be explained on the same basis as those produced by uniaxial crystals, the following explanation is tenable: In sections perpendicular to the optic axis of the crystalline system, if a cone of light passes through along this axis there will occur double refraction and retardation of peripheral rays due to the angulation of the rays. Because of this, isochromatic concentric rings of light and shadow are formed. Besides these rings there appear also in the interference figures of uniaxial crystals certain dark bars due to the relation of the differently oriented vibration directions (of the doubly refracted rays) to the principal planes of the Nicol prisms or polaroids. These curves of like vibration directions are termed "isogyres."

By a vectorial analysis it can be shown that all points on any radial line from the optic axis have the same intensity. This intensity is at its maximum on the line making an angle of 45 degrees with the vibration directions of the two polaroids and is equal to zero when parallel to them. As a result, there will be a gradation of light from the maximum intensity in the diagonal positions to darkness when the vibration planes are parallel to the principal sections of the Nicol prisms or polaroids. Thus the "cross" is formed. Conversely, if the polaroids are placed in parallel positions, the maximum amount of light will pass along the vibration plane, and instead of a dark cross there will be one of light. The greatest darkness in the interference figure will be on



A, front view of the anterior segment camera, with cover removed, showing attachment of the polarizing accessories: (1) polaroid inserted in illuminating beam and (2) polaroids attached to lenses. *B,* viewing ocular of the anterior segment camera showing inserted polaroid filter.

the diagonals. Strampelli demonstrated this phenomenon in the anterior segment also.

However, in the case of the cornea a more complicated figure is obtained. In addition to the aforementioned dark cross, alternating light and dark lines connecting the cross trees are seen at the limbus, giving a rhomboidal appearance to the figure. These could be attributed to the following factors: corneal curvature; varying thickness of the limbal portions, with respect to the central portions; varying lipin content, altering the mean refractive index, and mechanical factors associated with the ophthalmoscope, which lead to anomalous retardation and

birefringent effects. Furthermore, the image formation is governed by the constants of the cornea-aqueous refractive system. When one considers that the principal plane (reduced eye) lies 1.35 mm. deep to the corneal vertex, it may be deduced that the interference figure formed by a crossed polarized optical system lies in the vicinity of this principal plane.

In pursuance of my studies of polarization as it relates to the ocular structures, I designed certain polarizing attachments for the Zeiss anterior segment camera which facilitate the making of photographs of the anterior segment with crossed polarized or plane polarized light. The pictures may be made simultaneously or separately in monochrome or color film and permit analytic studies of the phenomena.

LESTER STEIN, M.D., Brooklyn.

SUBCONJUNCTIVAL HEMORRHAGE AS A SIGN OF DOUBLE PERFORATION OF GLOBE

To the Editor:—In the April issue of the ARCHIVES OF OPHTHALMOLOGY (**25:**672, 1941) there is an article by Friedman entitled "Subconjunctival Hemorrhage as a Sign of Double Perforation of the Globe by a Foreign Body."

The author stated that he first called attention to this symptom in 1931 (Subconjunctival Hemorrhage in Intraocular Foreign Bodies, ARCH. OPHTH. **6:** 919 [Dec.] 1931). He made no claim of priority for this observation, and he made no mention of its having been observed by others. It is of historic interest that the same observation was made by Rübel (Beitrag zur klinischen Diagnose der Doppelperforation des Bulbus durch Eisensplitter, *Klin. Monatsbl. f. Augenh.* **48:** 572, 1910). Rübel's article was abstracted in the Ophthalmic Year Book (Jackson, E.: The Ophthalmic Year Book, Chicago, 1911, vol. 8, p. 282).

Rübel regards subconjunctival hemorrhage which is not connected with the primary wound or ecchymosis of the lower lid as the surest test and commonest sign of double perforations. My own experience has been that if the symptom is present it is significant of a double perforation but that its absence does not exclude the existence of a double perforation.

W. ZENTMAYER, M.D., Philadelphia.

Obituaries

JOSEPH AUGUSTUS WHITE, M.D.
1848-1941

On Feb. 16, 1941, in the ninety-third year of his age and in full possession of his mental faculties, Dr. Joseph A. White, of Richmond, Va., whose name was well known in the ophthalmologic world and particulary in the South, passed to his reward.

Joseph A. White, the son of Ambrose A. White and Mary Hurley White, was born in Baltimore on April 19, 1848. His family was one of distinguished lineage, tracing its descent from colonial forbears long resident in Frederick County, Md.

As his parents were devout members of the Roman Catholic Church, his preliminary education was naturally acquired in the schools of that denomination. He attended preparatory schools in Baltimore, after which his collegiate training was obtained at Rock Hill College, Ellicott City, Md., Loyola College, Baltimore, and finally Mount St. Mary's College, Emmitsburg, Md., where he graduated with the degree of Master of Arts. Having early decided to study medicine, he then entered the medical department of the University of Maryland, from which he graduated in 1869.

He then pursued graduate studies for three years abroad, studying in turn at the École de Médecine, Paris, France, and at the Universities of Baden, Heidelberg and Berlin, Germany. As a student in Germany during the Franco-Prussian war he had an opportunity to see and study many of the casualties incident to that conflict.

Dr. White returned to the United States in 1872 and for a few years practiced general surgery in Baltimore. Having decided to limit his work to diseases of the eye, ear, nose and throat, he moved in 1879 to Richmond, Va., where he became a pioneer in this specialty and where he continued in active practice almost to the time of his death.

High intellectual endowments, together with a charming personality and adequate training for his special work, contributed immediately to his success, and he at once became a prominent figure in the professional and social life of his adopted city. In the early days of his practice there were comparatively few ophthalmologists and otolaryngologists in the South. In point of fact, the territory between Washington, D. C., and Atlanta, Ga., was still uninvaded, and Dr. White's skill was soon heralded far and wide. He therefore quickly acquired a large practice and attained a position of leadership in his special work throughout the South.

That Dr. White was held in high esteem by his colleagues is evident from the fact that he not only was elected to membership in practically all of the national ophthalmologic and otolaryngologic societies but was given positions of honor in many of them. In 1884 and 1885 he was chairman of the Section on Ophthalmology of the American Medical Association and was a past president of the Richmond Academy of Medicine, the Tri-State Medical Association of the Carolinas and Virginia, the Medical Society of Virginia and the American Laryngological, Rhinological and Otological Society.

He was one of the founders of the University College of Medicine of Richmond and occupied the chair of ophthalmology in that institution until it was merged with the Medical College of Virginia. He served in the same capacity in the latter for many years and on his retirement was made professor emeritus. He established and probably largely financed the Richmond Eye, Ear and Throat Infirmary, an institution operated exclusively for the care of indigent patients.

As a teacher he was held in high regard by his pupils. His alert mind, his dexterous hand, his bright and apt comments, his interesting and unique personality excited the admiration of those who had the privilege of being taught by him. He admirably succeeded in making ophthalmology interesting to the undergraduate—a difficult task for most teachers.

In the course of his career Dr. White was a frequent contributor to the literature on ophthalmology and otolaryngology. His papers were clear and concise and invariably covered well the subject presented. In discussion, while he had decided convictions of his own, he was tolerant of the opinions of others, and when he had occasion to criticize his comments were constructive and not caustic. His papers, though always concerned with some phase of his specialty, were invariably well received by general medical audiences.

No record of the professional attainments of Dr. White would be complete without reference to that engaging part of his personality which endeared him to a host of friends and associates. He possessed something besides scientific knowledge and skill. He was prominent in Richmond civic and social life for six decades; he was at one time president of the Virginia Society of the Sons of the American Revolution, as well as a member of the Westmoreland and Commonwealth Clubs, the Deep Run Hunt Club of Virginia and the Richmond German Club.

He had a peculiarly youthful outlook that led young men and young women to seek his companionship. Though he lived far beyond the allotted three score and ten years, he never grew old. Until a few years ago this aged man was an active horseman. He rode not only

on the roads but to hounds and in the show ring. He more than once suffered nasty spills and broken ribs, but these mishaps did not deter him from future efforts, and at the age of 77 he was awarded a cup for jumpers at the Deep Run Hunt Club.

Dr. White was fond of dancing, and, as a member of the Richmond German Club, nothing gave him greater delight than attending the dances in honor of the Richmond debutantes as they were formally

JOSEPH A. WHITE, M.D.
1848-1941

presented to society. His small, wiry, graceful figure was a familiar sight on these occasions. So expert was he in the art of dancing that it was said of him: "He never trod on the toes of a tremulous debutante."

In his younger days the game of golf probably did not appeal to him as strongly as did some of the more active outdoor sports. At the age of 65, however, he became interested in the game, and he continued to play with enthusiasm until he was 85.

For more than half a century he occupied a unique position in the social life of Richmond. A man of vigorous intellect, kindly humor and charming social gifts, he was always a center of attraction. The warmth and cordiality of his greeting, the convivial and ingratiating influence of his manner and his quality of good fellowship for all ages made him known and beloved by persons in all stations of life. He was ever ready with a story and was ever ready to listen—but not for long.

On his seventy-ninth birthday many of his distinguished ophthalmologic colleagues, headed by Dr. de Schweinitz, together with a large group of medical friends from other states and cities, gathered in Richmond to honor Dr. White and to pay tribute to his accomplishments as a pioneer in the field of ophthalmology in Virginia and the South. The event was a notable one in the medical history of Richmond and was a fitting tribute to the man who, through a long period of years, had made valuable contributions to the progress of ophthalmology throughout the South and the nation.

Dr. White married the former Sophie Berney, of Montgomery, Ala., who died in 1901. Surviving him are one daughter, Mrs. Roger Pryor Walker, of New York, and several grandchildren.

HUNTER H. MCGUIRE.

MORITZ von ROHR

1868-1940

The days of our years are threescore
and ten . . . . .

Psalm 90: 10.

This quotation may well be applied to Moritz von Rohr, who despite his more or less constant suffering from tuberculosis of the bones over a period of many years continued for forty years to carry on at the Zeiss Works, first as assistant to Dr. Rudolph and later as active manager and collaborator. His death, on June 6, 1940, terminated a long and useful career.

For a more detailed report of his life and an outline of his writings, the reader may refer to the article by Boegehold,[1] friend and collaborator of von Rohr, in which he credits him with creating and elaborating many of the optical devices put out by the Zeiss Works and with publishing about five hundred papers, many of them of basic importance in lens optics.

Moritz von Rohr was a prodigious worker, a constant delver into old archives, dust covered and half forgotten, constantly searching and sifting the important truths from the nonessentials. His favorite occupation, to which he devoted himself whole heartedly and assiduously after his retirement as an active official of the Zeiss Works, in 1935, was historical research, and many of his papers testify to his ability in this field.

In the biographic sketch, Boegehold mentioned the most important of his works the seriousness and painstaking devotion with which he attacked his problems, his skill in grasping and disseminating his knowledge, and his humanism, love of justice and loyalty—outstanding characteristics of an outstanding personality.

In the death of Moritz von Rohr the world has lost not only a great scientist but a great man.

CAMILLA HÜBSCHER.

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1. Boegehold, H.: Ztschr. f. ophth. Optik. **28**:97-101, 1940.

HERBERT WRIGHT WOOTTON, M.D.
1867–1941

The members of the Section of Ophthalmology of the New York Academy of Medicine record with sympathy the death of one of their oldest fellows, Dr. Herbert Wright Wootton. Dr. Wootton became a member of the New York Academy in 1896, and until his retirement five years ago he was an energetic participant in its activities. He served as secretary to the Section of Ophthalmology from 1906 to 1909 and as chairman from 1910 to 1912.

Dr. Wootton died on February 28, after a brief illness, at his home in Old Lyme, Conn. He was in his seventy-fourth year.

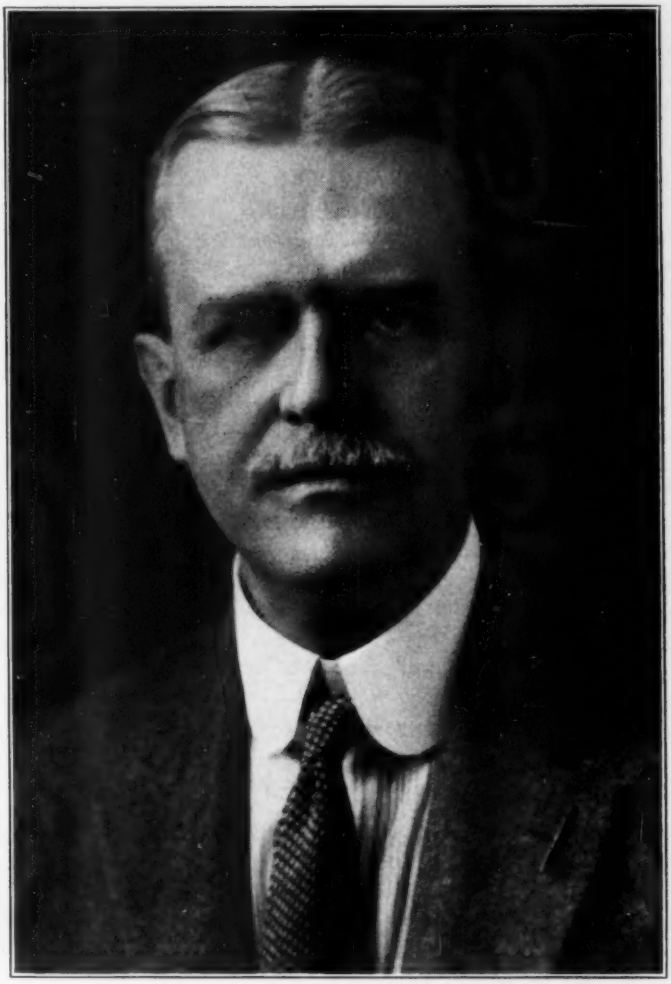
He was born on Dec. 25, 1867, in the city of New York, son of Edward Henry and grandson of General Horatio G. Wright of Civil War fame, for whom Fort Wright on Fishers Island, N. Y., is named. After preparatory studies at the College of the City of New York, he entered the Columbia University College of Physicians and Surgeons, from which he received his medical degree in 1888. He won the Harsen Prize Medal, which was awarded to the member of the graduating class attaining the greatest proficiency in all branches. His career in ophthalmology began in 1898, when he became a clinical assistant at the Manhattan Eye, Ear, and Throat Hospital. In 1899 he was made an assistant surgeon, in 1910 a junior surgeon and in 1914 a surgeon. He was promoted to surgeon director in 1924. He served as secretary to the board of surgeons from 1919 to his retirement, in 1932. For twenty-one years he was a member of the New York Board of Health, for ten years serving as diagnostician for contagious diseases, later devoting much of his energy to the improvement of ocular hygiene in school children and the study of trachoma.

He served as visiting ophthalmologist at Gouverneur, Willard Parker and United States Marine hospitals and on Draft Board 150 during the World War. He was a member of the New York county and state medical societies, the New York Ophthalmological Society, of which he was a president, and the American Ophthalmological Society.

His clubs included Columbia University, Quill and the Loyal Legion. He was beloved by his staff and the residents of the Manhattan Eye, Ear, and Throat Hospital, to whom he was a walking encyclopedia. One of his greatest interests was teaching, and he spent many years of his life in postgraduate instruction. Although he was not a prolific writer, he made many contributions to ophthalmologic literature, all of which were significant.

Dr. Wootton was an accomplished linguist; he spoke German, French and Italian fluently, and one of his hobbies was reading rare

Latin editions. He was an Episcopalian and a staunch Democrat. It was his firm conviction that in a democratic country like the United States the inhabitants received, locally and nationally, the kind of governing they deserved.



HERBERT WRIGHT WOOTTON, M.D.
1867–1941

He married Florence Randolph Sanders. Surviving are a son, Edward Wright Wootton, of Alexandria, Va., and a daughter, Anne (Mrs. F. B. Hillhouse), of Summit, N. J.

DAVID H. WEBSTER, M.D., New York.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE INFLUENCE OF THE TUNICA VASCULOSA LENTIS UPON THE DEVELOPMENT OF CYSTS DERIVED FROM THE PIGMENT EPITHELIUM. E. PURTSCHER, Arch. f. Ophth. **140:** 569, 1940.

The microphthalmic right eye of a 12 year old girl (case 1) showed, in addition to a coloboma of the choroid, a brown cyst occupying the pupillary area. After an iridectomy, which included partial removal of the cyst, it could be seen that the lens was dislocated downward and that the ciliary processes above (in the region of the coloboma) were markedly lengthened and, in the periphery, merged into a continuous membrane. Histologic examination of another microphthalmic eye (case 2) with an ectopic lens and a coloboma of the uvea revealed a large cyst lined by pigment epithelium and located in the posterior chamber. Thus the clinical observations in case 1 resembled in many respects the anatomic observations in case 2. Further study of case 2 made it seem probable that in the development of the cyst early extensive adhesions between the pigment epithelium and the tunica vasculosa lentis and disturbed involution of the latter had been important factors. The unusual length of the ciliary processes was thought to be the product of growth under abnormal conditions and not just the result of stretching.

P. C. KRONFELD.

Bacteriology and Serology

THE OCULAR SHWARTZMAN PHENOMENON. T. E. SANDERS, Am. J. Ophth. **22:** 1071 (Oct.) 1939.

Sanders discusses the Shwartzman phenomenon. Shwartzman found that if a potent bacterial filtrate is injected into the skin of a rabbit no appreciable reaction results, but if twenty-four hours later the rabbit is given an intravenous injection of the same or another potent filtrate there appears after four or five hours extremely severe hemorrhagic necrosis at the site of the local injection. Sanders reviews the literature and notes that this phenomenon occurred consistently in the bulbar conjunctiva but did not so occur in the cornea. The results obtained by different investigators have varied as to its occurrence in the anterior chamber and in the vitreous. Sanders repeated the experiments on rabbits and in general confirmed the previous observations. He suggests that this phenomenon may explain the action of a focus of infection and some phases of the foreign protein reaction.

W. S. REESE.

A Study of Methemoglobin-Producing Organisms in Ocular Inflammations. M. A. Wood, Am. J. Ophth. **22:** 1111 (Oct.) 1939.

After a bacteriologic study of all patients with conjunctival and corneal disease and all patients awaiting operative treatment regardless of the condition of the conjunctiva, Wood draws the following conclusions:

"1. The incidence of Str. viridans and D. pneumoniae in ocular inflammations is quite similar.

"2. The criteria for the diagnosis of D. pneumoniae in order of importance are:

"a. Reaction to type-specific antisera,

"b. Bile solubility,

"c. Colony characteristics upon solid and liquid culture media,

"d. Microscopic characteristics after growth in broth media.

"3. Inulin fermentation alone is of no value in differentiating D. pneumoniae from Str. viridans.

"4. In order of frequency among pneumococci, type VII (25 per cent) was most common, followed in order by type XIV (12 per cent) and type XXIII (10 per cent). The remaining types were less frequently encountered."

W. S. Reese.

Conjunctiva

Tuberculosis of the Conjunctiva. N. K. Ivanov, Vestnik oftal. **14:** 24, 1939.

Since tuberculosis of the conjunctiva is rare, 3 cases of this condition, of varying clinical courses, are reported. The patients were girls aged 12, 14 and 15 years respectively.

In 1 case the primary lesion of tuberculosis was diagnosed as a chalazion, but later it took the course of a typical tuberculous ulcer, with enlargement of the regional glands. In the second case tuberculosis of the conjunctiva was metastatic, with no enlargement of the glands; its course was torpid, and the lesions resembled the follicles of trachoma. In the third case, in which the involvement was severe, the lesion resembled a tumor of the conjunctiva, but histopathologic examination revealed its tuberculous character. It has spread from the nasal and oral cavities to the conjunctiva. The course was progressive, the conjunctival lesion spreading to the cornea. There was much granulation tissue. The final result was atrophy of the eyeball. In the first case therapy for tuberculosis gave good results, while in the other cases roentgen therapy was used with no effective results.

Ivanov discusses the ways of infection and the clinical picture of tuberculosis of the conjunctiva. He believes that the variety of the clinical forms of tuberculosis of the conjunctiva can be explained by different reactions of the organism to the tubercle bacilli, by the different immunobiologic conditions and by the stage of the inflammatory process.

O. Sitchevska.

Congenital Anomalies

HUMAN CYCLOPIA WITH ASSOCIATED OCULAR ANOMALIES. I. E. WILBER, Am. J. Ophth. **22**: 1120 (Oct.) 1939.

Wilber reports a case of cyclopia in an 8 month premature girl who lived for one-half hour after birth. The mother was a normal primipara. The gross and microscopic observations are enumerated, and an interpretation is given from a morphologic and chronologic standpoint, followed by a discussion of the cause of cyclopia. The following summary is given:

"A case of cyclopia, type synophthalmia bilentica, with concurrent ocular anomalies has been described and its embryological development delineated. Modern theories of the teratogenesis of cyclopia have been discussed."

W. S. REESE.

Cornea and Sclera

EPISCLERITIS AND SCLERITIS, WITH ESPECIAL CONSIDERATION OF EPISCLERITIS PERIODICA FUGA: REPORT OF CASES. R. SEEFELDER, Klin. Monatsbl. f. Augenh. **103**: 417 (Oct.-Nov.) 1939.

The author observed a woman aged 40, with episcleritis periodica fugax for three and one-half years after March 1936. She had suffered attacks in both eyes every two to three weeks since 1928. These attacks, lasting for about one week, were very painful and were accompanied with general malaise. Rheumatic, arthritic and menstrual influences were ruled out. The cause, in Seefelder's opinion, may have been an abnormal sensitivity of the episcleral tissue, which responded with an inflammation to divers nonspecific excitations. Amelioration of the attacks and longer intervals between them (one such interval lasting six months) were obtained by intramuscular injections of calcium. The same therapy yielded a similar result in a man aged 35 and produced decided improvement in a woman aged 40, who is still under treatment. In the fourth case, that of a woman aged 60, episcleritis was complicated by recurrent rheumatic iritis. The severity of the condition increased, finally requiring enucleation of one eye after an iridectomy had been done for occlusion of the pupil a few months previously. Histologic examination showed extensive destruction of a portion of the ciliary body, giant cells and other tuberculous changes. The Wassermann reaction was negative. This case, in the author's opinion, is proof of the tuberculous causation of episcleritis in some cases as pointed out by several writers. Seefelder suspects that episcleritis may be secondary in some cases of moderate primary disease of the anterior portion of the uvea.

K. L. STOLL.

CIRCUMSCRIBED SCLEROMALACIA IN ADVANCED AGE: REPORT OF CASES. W. KYRIELEIS, Klin. Monatsbl. f. Augenh. **103**: 441 (Oct.-Nov.) 1939.

In an introductory chapter the author refers to the bluish discoloration in the sclera at the insertion of the horizontal ocular muscles and to similar discolorations resulting from deep inflammation of the

episcleral tissue. He then mentions the related diseases scleromalacia perforans (Van der Hoefe) and scleritis necroticans (Rochat).

Three cases of the author's own observation are reported. The patients were men, aged 77, 70 and 60 respectively. There were well defined atrophic tissue changes in the sclera, especially in the outer layers; they were located at the insertion of the horizontal rectus muscles, within the area of the palpebral fissure. Histologic examination of one of the eyes showed massive calcification in the sclera, aside from atrophy of its superficial layers. Personal observations and references found in the literature indicate that these defects are furthered by rheumatic disease and that inferiority of the tissues influences the development of chronic rheumatism as well as the scleral degeneration. Senile disorders of the metabolism and of the blood vessels appear to be important. It is possible that disturbances of metabolism and blood vessels per se may suffice to produce atrophy of the sclera.

K. L. STOLL.

Experimental Pathology

ELECTROCHEMICAL PROCESSES UNDERLYING CHALKOSIS OF LENS AND CORNEA. S. MIELKE, Arch. f. Ophth. **141:** 644, 1940.

If a copper-containing foreign body is retained in the eye impregnation with copper salts occurs, as far as the clinician can tell, in the cornea, lens and retina. In the cornea the copper salts are deposited between the endothelium and Descemet's membrane, and in the lens, between the capsule and the epithelium in the pupillary zone. This selective distribution of the copper salts in the eye may, according to the author, be produced by the constant electric current which is present in every vertebrate eye and which renders the cornea electropositive as compared to the posterior pole of the eye (demarcation current). In order to prove this concept experimentally the author introduced a piece of copper wire through the posterior pole into the vitreous of a pig's eye and connected it with the positive pole of a battery. The cornea of this eye was immersed in saline solution which, through a carbon electrode, was connected with the negative pole of the battery. Thus a galvanic current was sent through the eye to intensify the natural demarcation current. The result was a very rapid impregnation of the cornea and the lens with copper, which in all essential points closely resembled the chalkosis that occurs in human eyes containing a piece of copper. Electrochemical considerations thus provide a plausible explanation of the peculiar distribution of copper in the eye.

P. C. KRONFELD.

General Diseases

DYSKEPHALY (DYSOSTOSIS CRANIOFACIALIS, CROUZON'S DISEASE) IN TWO SISTERS: REPORT OF CASES. H. SCHLÄPFER, Klin. Monatsbl. f. Augenh. **103:** 469 (Oct.-Nov.) 1939.

In 1912 Crouzon described a hereditary disease which he called dysostosis craniofacialis, the main symptoms of which were deformations of the skull and facial bones and ocular disturbances, such as exophthal-

mos, divergent strabismus and amblyopia. Vogt added another symptom, insufficient development of the intermaxillary bone. He stressed such ocular symptoms as wide interocular distance, excessive exophthalmos due to increased intracranial tension, divergent strabismus, choked disk and atrophy of the optic nerve.

Schläpfer reports the cases of 2 sisters, aged 31 and 23 respectively, with the aforementioned symptoms. Two other sisters and 1 brother showed normal development of the skull; another brother, merely prognathism and a very high palate. The roentgenograms furnish proof of early synostosis of the cranial sutures and widening of the base of the skull, causing tower skull in 1 sister and occipital widening of the skull in the other. Secondary widening of the sella turcica was noted in both sisters and hypoplasia of the intermaxillary bone in 1.

K. L. STOLL.

Injuries

OPHTHALMIC INJURIES IN WAR. N. I. SHIMKIN, Brit. J. Ophth. **24:** 265 (June) 1940.

The observations on which Shimkin bases his summary were made at the chief casualty-clearing station at the Russian southwestern front during 1914 and 1915, and from 1916 during his experience as consulting ophthalmologist to the army on the same front.

1. The author gives the results of his observations of 238 cases of ocular injuries at the chief casualty-clearing station and of over 593 cases in base hospitals. He illustrates some of them by histories of the injuries and by photographs and drawings of those types of ocular injuries not met in times of peace. He discusses the difficulties in diagnosis, treatment and prognosis for the sake of those oculists who did not take part in the last war.

2. He examines the character of the explosive action of bullets or other gun projectiles in wounding the orbit and stresses the enormous kinetic energy developed. He points out that the real destruction of the orbit and its contents is much more extensive than is observed on examination of the external wound. All orbital wounds in which the missile passes over the arcus zygomaticus should be considered cranial injuries. By these injuries the upper walls of the orbit, the meninges and the brain are wounded.

3. On the basis of numerous ophthalmoscopic and clinical observations he considers that concussion of the eye and concussion of the brain are nothing but disturbance of the vascular system in these parts. The severity of the concussion is in direct proportion to this disturbance, which may result in varying degrees of degeneration of the nerve tissue of the retina and of the cortical visual centers.

4. Among the 100 wounded there were 2.25 with ocular injuries and 20.4 with injuries of the head. Rifle bullets caused two thirds of all of the orbital and of the ocular injuries penetrating through and through. One third of such wounds were inflicted by artillery fire. A patient suffering from an ocular wound stayed in a hospital on an average of forty-six and eight-tenths days.

The ocular injuries in war are accompanied with dense adhesions of the sclera and the destroyed choroid to the underlying tissue, which greatly increases the difficulty of enucleation of the globe; therefore, the author suggests that patients with such injuries should be sent to a center where there is an oculist. The fear of sympathetic ophthalmitis is exaggerated; the author has observed only 1 example of irritation of the sympathetic nervous system, which occurred on the twenty-sixth day after the patient had been wounded.

6. Of 100 cases of ocular injuries, an eye had to be enucleated in 50. Of 100 men with ocular injuries and concussion, 77 lost the sight in one eye and 11.8 per cent that of both eyes. The present war threatens to produce a still greater number of blind men than did the last war, as in trench warfare the head, face and eyes are more often wounded than in "open warfare."

The article is illustrated.

W. ZENTMAYER.

Lacrimal Apparatus

BILATERAL GUMMATOUS SYPHILIS OF THE TEAR GLAND: REPORT OF A CASE. F. W. MEYER, Klin. Monatsbl. f. Augenh. **103: 200** (Aug.) 1939.

A woman aged 71 complained of diplopia and spontaneous swelling of the upper lid which she had noticed for three months prior to observation. The tumor, deviating the eyeball downward and to the nasal side, was suspected to be a mixed neoplasm of the lacrimal gland. Histologic examination revealed that it was doubtlessly syphilitic. Necrosis was predominant; caseous degeneration involved the central portions; perivascular infiltration with round cells and fibroblasts abounded, and epithelioid cells with necrosis, giant cells and endarteritis were noted. Filamentous keratitis developed after removal of the lacrimal gland. The author draws attention to the extreme rarity of bilateral syphilis of the lacrimal gland and to the fact that the Wassermann reaction was negative.

K. L. STOLL.

Lens

PREPARATORY STUDIES IN MEDICAL THERAPY OF CATARACT. H. K. MÜLLER, H. SOHR and A. BRÜNING, Arch. f. Ophth. **141: 621**, 1940.

The object of the studies reported was to find the most effective method of increasing in the aqueous of rabbits the concentrations of three substances known to be essential for the metabolism of the lens, namely, dextrose, glutathione and ascorbic acid. In the case of dextrose and of glutathione local application of a concentrated solution in the form of an eye bath was most effective, especially if combined with administration of a local anesthetic, such as pontocaine hydrochloride. The most pronounced and lasting elevation of the ascorbic acid content of the aqueous was produced by intravenous injection of the substance.

P. C. KRONFELD.

Lids

MYCETOMA OF THE EYELID. J. S. ALDRIDGE and R. KIRK, Brit. J. Ophth. **24:** 211 (May) 1940.

The patient was a Nubian girl aged approximately 4 years. A tumor had begun to develop in the upper eyelid at the outer angle of the right eye two months before and had attained the size of a marble. Sections of the tumor showed the morbid histologic appearance commonly associated with a fungous infection—a chronic nonspecific inflammatory lesion characterized by hyaline fibrosis, plasma cell and leukocytic infiltration, with occasional small imperfect giant cell formations. In the more densely infiltrated parts fungous grains could be seen irregularly distributed. Little in the way of detailed structure could be made out in the grains, and cultures were out of the question, but their appearance and relation to the associated histopathologic changes are sufficiently typical to establish a diagnosis of mycetoma. The authors could find but 2 other cases of such ocular involvement in the literature. In 1 of these cases the patient came from approximately the same region of northern Sudan as did the patient in the present case.

The article is accompanied with illustrations. W. ZENTMAYER.

Neurology

INVOLVEMENT OF THE OPTIC NERVE IN ENCEPHALOMYELITIS AFTER VACCINATION: REPORT OF CASES. F. VON HERRENSCHWAND, Klin. Monatsbl. f. Augenh. **102:** 815 (June) 1939.

This paper was prompted by Scheyhing's publication of a case of optic neuritis with transient blindness associated with meningoencephalitis following vaccination for smallpox. The author describes 3 cases of his own. The patients were boys aged 7, 7 and 8 years respectively. The bioscopic reports, illustrated by photomicrographs, and brief descriptions of the obduction are added. The ocular disease was limited in all but 1 case to the dural sheath of the optic nerve, sparing this nerve and also the septums between the bundles of its fibers. The process was secondary, in the author's opinion, descending from the meninges. Small foci were observed in the optic nerve of 1 patient resembling those found in the white cerebral substance in cases of encephalomyelitis following vaccination. This type of retrobulbar neuritis occurs, furthermore, in association with multiple sclerosis and lethargic encephalitis.

A point of histologic differentiation from these diseases, however, is the fact that glial proliferations forming continuous seams around the veins predominate in cases of encephalitis following vaccination.

K. L. STOLL.

OCULAR SYMPTOMS IN PATIENTS WITH TUMORS OF THE HYPOPHYSIS OBSERVED AT THE ZURICH OPHTHALMIC CLINIC AND THE ROENTGEN INSTITUTE, 1924 TO 1939: REPORT OF CASES. P. KARBACHER and H. R. SCHINZ, Klin. Monatsbl. f. Augenh. **103:** 541 (Oct.-Nov.) 1939.

The authors report on 27 living patients given radiation therapy for tumors of the hypophysis during the last fifteen years. The syn-

dromes were clinically divided into four groups; (1) the Cushing type, obesity with basophilic adenoma; (2) the Pierre-Marie type, acromegaly with eosinophilic adenoma; (3) the Fröhlich type, dystrophia adiposo-genitalis, and (4) the type of sellar tumor which is not associated with definite endocrine symptoms. Twelve cases are reported in detail, and the tables record data relative to treatment, field of vision, Bjerrum's screen, vision, appearance of the disks and divers symptoms; schemes of the field of vision and of the screen are added. Roentgen ray cataract was not observed in a single instance. The question of whether to operate or to employ irradiation is discussed. Irradiation, in the form of protracted fractional application, is the only therapy for recurrences. Operation is insufficient for complete eradication of tumors, although it may greatly relieve the patient and will furnish histologic evidence; operative intervention must be contemplated as soon as the inefficiency of irradiation alone has become evident. Operation should be resorted to if the ocular symptoms remain unimproved after a few weeks or if aggravation of the ocular symptoms occurs during the radiation therapy.

K. L. Stoll.

Ocular Muscles

A New Surgical Method of Correcting Paralytic Strabismus with Contraction of the Antagonist: Report of Cases. A. Vogt, Klin. Monatsbl. f. Augenh. **103:** 296 (Sept.) 1939.

The tendon of the contracting muscle is thoroughly severed from the eyeball with correction of the position of the deviating eye. Then a double-armed suture is tied around both (intact) tendons of the rectus muscles, which act vertically to the muscle which has been detached. The sutures are pulled tight, and after being carried from inside the lids through the skin they are fastened over gauze coils. A cut illustrating the operation and pictures demonstrating the results in 2 cases are added. The paralysis of the abducens nerve in one of these cases was of thirty years' standing.

K. L. Stoll.

Operations

The Extraction of Nonmagnetic Intraocular Foreign Bodies. F. K. Leydhecker, Arch. f. Ophth. **141:** 665, 1940.

The various methods for the diagnosis and localization of intraocular nonmetallic foreign bodies are thoroughly reviewed. Comberg's roentgen method of localization consists of the taking of one postero-anterior and one lateral view of the injured eye, using a contact glass with lead marks. During the first picture the patient's head rests with the chin and nose on the film carrier, the visual axis of the eye being in the vertical position and perpendicular to the film. The lateral view is taken with the patient sitting up and the visual axis in the horizontal position. Leydhecker's objection to Comberg's method is that the location of the foreign body within the eye may not be the same in the two positions of the head (and eye) in which the two pictures are taken. He therefore takes the two views by changing the position of the x-ray tube and film and without changing the position of the patient's

head. The patient lies on a stretcher with his head suspended in the horizontal position by radiotranslucent straps. During the taking of the posteroanterior view the tube is below the patient's head while the film carrier rests on his face. During the taking of the lateral view the tube is on the side of the uninjured eye and the film carrier on the side of the injured eye.

During the extraction, observation of the position and action of the forceps on a two plane fluoroscopic screen is recommended. For the removal of nonmagnetic foreign bodies, which are difficult to demonstrate roentgenologically, the author has devised an instrument on the order of a cystoscope combined with a forceps. This instrument is similar to the one recommended by H. E. Thorpe (ARCH. OPHTH. **15:** 308, 1936).

The comprehensive bibliography (13 pages of small print) and the excellent illustrations add greatly to the value of the paper.

P. C. KRONFELD.

Parasites

SUBRETINAL CYSTICERCUS IN THE MACULAR REGION. A. KRUTOVA, Vestnik oftal. **17:** 146, 1940.

A man aged 52 became suddenly blind in the left eye. He was sent to Moscow with a diagnosis of a hemorrhage in the macular region. The diagnosis was changed to subretinal cysticercus in the macular region. A round bluish white vesicle measuring about 3 disk diameters was seen in the macular region, in the center of which the head of the parasite was seen. The wavy motion of the vesicle could be seen with Gulstrand's ophthalmoscope. The blood count showed moderate eosinophilia, and Ascaris ova were found in the stools.

Operation first consisted in canthoplasty and division of the external rectus muscle, but the posterior pole of the eyeball could not be reached, so that Krönlein's resection of the orbit was resorted to by Strakhov (who first suggested this operation for the removal of cysticerci from the posterior pole of the eye). An incision 2.5 to 3 mm. long was made in the sclera, through which the cysticercus came out with its walls intact, with no loss of vitreous. Recovery was uneventful, and the patient could count fingers at 3 meters soon after the operation.

O. SITCHEVSKA.

Pharmacology

THE PRACTICAL USE OF HOMATROPINE-BENZEDRINE CYCLOPLEGIA. L. S. POWELL, Am. J. Ophth. **22:** 956 (Sept.) 1939.

After a study of 100 cases, Powell draws the following conclusions: "Homatropine and benzedrine used in combination will produce in 60 minutes complete practical cycloplegia in a high percentage of patients between the ages of 16 and 31 years. Beginning recovery is evident in four hours. Eserine-salicylate solution, 1 per cent and 0.5 per cent buffered to be isotonic with tears, will overcome homatropine-benzedrine cycloplegia promptly and restore the power of accommodation within one-half hour."

W. S. REESE.

The Pupil

PUPIL AND MIDBRAIN. W. R. HESS, Klin. Monatsbl. f. Augenh. **103:** 407 (Oct.-Nov.) 1939.

Hess publishes the result of his research on the interrelation of the vegetative (autonomic) and animal (cerebrospinal) functions and on the important role played by the ocular symptoms in this process. Two methods are at hand to investigate the function of the midbrain in connection with the functions of the entire central nervous system and the methods of excitation and elimination. The former (excitation) is elicited with an electric object with which one portion of the midbrain after the other is touched under constant control of the pupil. The author inserts very fine electrodes through small holes drilled into the cranium of the animal. The elimination is performed by electrocoagulation with diathermic current. The symptoms are photographed with a moving picture film, which allows exact study of the pictures showing the anatomic changes in the brain of the killed animal. The zone for mydriatic actions has been found in the posterior portion of the hypothalamus and its transition into the midbrain, and the area from which miosis is produced was found in the anterior portion of the hypothalamus. A second miotic zone was located in the medial portions of the ventral nuclei of the middle part of the thalamus. The miotic effect produced in this zone is especially intense, requiring the application of 4 volts to enlarge the pupil again in some cases. The thalamus and the hypothalamus possess a field in which simultaneous narrowing of the pupil and of the palpebral fissure may be obtained. Hess refers to the clinical necessity of controlling the relation between the size of the pupil and absolute, standardized illumination in cases of suspected central lesions. In this connection it is important to watch for concomitant symptoms of ergotropic (sympathicotonic) and trophotropic (parasympathicotonic) functions.

K. L. STOLL.

Physiology

THE CENTRAL VISUAL SYSTEM: EVIDENCE AGAINST BILATERAL REPRESENTATION THROUGH THE SPLENIUM OF THE CORPUS CALLOSUM. O. R. HYNDMAN, Arch. Neurol. & Psychiat. **42:** 735 (Oct.) 1939.

No case has been reported in which from a clinical standpoint incontrovertible evidence has been furnished for or against a possible role of the corpus callosum in central vision. The author believes that the case reported furnishes that evidence.

Full macular vision was retained after resection of the right occipital lobe to the tip of the splenium of the corpus callosum, in addition to section of the corpus callosum to a point 2¼ inches (5.7 cm.) from the posterior tip. Consequently, the splenium of the corpus callosum plays no role in retention of central vision.

Central vision is probably (at least in part) represented anterior to the accepted visual cortex and is probably not represented bilaterally.

R. IRVINE.

Retina and Optic Nerve

INTERMITTENT OCCLUSION OF THE CENTRAL RETINAL ARTERY. T. E. SANDERS, Am. J. Ophth. **22:** 861 (Aug.) 1939.

Sanders reports the case of a man aged 60 in whom complete loss of vision in the left eye occurred after marked exertion. This was promptly relieved by rest, the loss being for very short periods, a few times persisting for ten minutes. Ocular examination gave essentially negative results, but efforts to reproduce the loss of vision were finally successful, and after marked exertion the ophthalmoscope revealed all the retinal arteries reduced to thin white threads. The veins seemed normal. The disk was pale and the retina hazy. After disappearance of the occlusion there were usually dilatation and distention of the retinal veins for ten or fifteen minutes. The patient was given a thorough physical examination and tested in different ways to determine the cause of the retinal occlusion. It was found that this occurred when the blood pressure reached 180 and that it failed to take place after the use of vasodilators, such as glyceryl trinitrate. Increased intracranial pressure, hyperventilation and posture were ruled out as causative. The author gives the following summary:

"1. A case of intermittent occlusion of the central retinal artery repeatedly observed ophthalmoscopically is reported.

2. Fundus photographs taken before, during, and after an attack of occlusion are discussed.

3. The causative factors of endarteritis and arterial spasm are evaluated."

W. S. REESE.

NEURITIS PAPULOSA. A. FUCHS and P. SATANOWSKY, Arch. de oftal. de Buenos Aires **14:** 661 (Sept.) 1939.

The authors report a case in which after an injury the disk margins became blurred and a pink exudate appeared in the center of the disk. Above and outward from the disk a cicatrized choroiditic lesion was observed, and near it was another cotton-like focus, apparently in the external layers of the retina. A thin exudate joined the latter focus with the one on the disk. Vision was reduced to one third. The visual field showed corresponding defects. The Wassermann and Kahn serologic reactions were negative. Improvement with cicatrization of the chorioretinal focus and increase of vision to two thirds was obtained after twenty-five injections of mercury bicyanide. The exudate joining the papillary with the choroidal focus was absorbed. The final aspect was that of a case of Jensen's neuritic choroiditis juxtapapillaris. Reference is made to a similar case observed by the author and to one observed by Kuriat.

C. F. FINLAY.

INCREASE OF VISION OF THE DAMAGED RETINA: REPORT OF CASES. G. GUIST, Klin. Monatsbl. f. Augenh. **103:** 77 (July) 1939.

Interruption of the circulation of blood causes death of the retinal nerve cells after fifteen to thirty minutes. Preservation of vision weeks after an embolism has lodged in the central retinal artery is possible

only in cases in which the embolism does not occlude the artery completely. After discussing this topic in detail, the author records the history of about 8 patients in whom he succeeded in increasing vision after the retina had been damaged by a number of chorioretinal diseases. These included disciform degeneration of the retina (Kuhnt-Junius), retinitis proliferans and drusen of the glassy lamella of the retina. He used "xipoid retinale," which is prepared from retinal tissue. It is free from hormonal admixture. The contents of one ampule, containing 1.1 cc. of this preparation, are injected into the skin at two spots on the upper part of the arm, about 4 cm. distant from one another. Increase of vision was observed after two to three hours in some cases and after one day in others. The effect lasted three to five days. The injections therefore must be repeated for some length of time; they yield increasing results. This therapy, in all appearance symptomatic, may prove to attack the cause of some of the diseases treated.

K. L. Stoll.

Lesions of the Optic Nerve Produced by Vitamin Deficiency. A. Pillat, Klin. Monatsbl. f. Augenh. **103**: 97 (July) 1939.

A Spanish oculist reported on a typical ocular syndrome observed after cessation of the Spanish Civil War in nationalistic soldiers who had been kept prisoners by the marxist (communist) army. He noted in each of these soldiers xerosis of the conjunctiva, temporal pallor of both disks, central scotoma with a normal periphery of the field of vision and reduced vision which could not be brought up to the normal standard after correction of ametropias. The other intraocular organs were observed to be normal. The condition resembled retrobulbar neurosis, and in the writer's opinion it was caused by avitaminosis. These soldiers received for daily rations only scant food consisting of boiled rice and lentils. Pillat stated that he observed similar conditions in China, that Japanese physicians drew attention to these cases and that avitaminosis (A, B and B_1) was undoubtedly the cause of this disturbance. Chinese, Japanese and Argentine publications in point are cited.

K. L. Stoll.

Trachoma

Research on the Etiology of Trachoma. A. Santoni, Ann. di ottal. e clin. ocul. **68**: 32 (Jan.) 1940.

The author reviews the literature concerning the infectivity of trachoma and the records of positive results from human and animal inoculations. He reports his own attempts to grow the trachoma virus in tissue culture. He employed as a medium layers of epithelium removed from the human cornea of stillborn fetuses, since by this means a pure culture of epithelium was obtained, without connective tissue cells. The epithelial explants were floated on heparinized human plasma with the addition of embryonal chicken and human splenic extract. The trachomatous material was obtained from subjects showing inclusion bodies in epithelial scrapings. Inoculation was made after the tissue culture had grown several days. Unfiltered material was employed and also material passed through a collodion membrane of 0.7 micron pore

size. Cultures free of secondary infections were obtained six times after inoculation with trachomatous material. These were allowed to grow three to four days and then transferred to a second culture, while others were used for histologic examination. The results were negative.

Nothing suggesting inclusion bodies was found in the tissue inoculated with trachoma which differed from that in the uninoculated tissue cultures.

S. R. GIFFORD.

Tumors

TUMORS OF THE ORBIT. A. POKROWSKY, Vestnik oftal. **15:** 11, 1939.

This is one of a series of articles on tumors of the orbit; (1) traumatic epithelial cysts and (2) simple lymphomas or lymphadenomas of the orbit. Pokrowsky reports in detail a case of a tumor of the orbit which resembled clinically a dermoid, because the cyst contained a fatlike substance with a number of hairs. Ten years previously the patient had fallen and had hit the upper edge of the right orbit. The direct connection of the growth with the site of trauma indicated that the source of the tumor was the epithelial tissue of the skin, which, together with a bone fragment, penetrated the orbit. The tumor differed from a dermoid cyst in the presence of large diverticula and side channels. The epithelial cyst should be removed early because of pressure on the surrounding tissues.

The clinical and histopathologic picture of a lymphoma of the right orbit is reported. Pokrowsky discusses the difficulty of diagnosing orbital tumors and gives the points of differentiation between lymphoma, lymphosarcoma and leukemic lymphadenitis. The lymphoid tissue of the lacrimal gland frequently may be the source of orbital lymphoma, although there may be "lymphatic metaplasia" in the loose connective and fatty tissue of the orbit. An early removal of orbital lymphoma is urged because of the possibility of malignancy later. A number of case reports are cited from the literature, and various theories on the origin and nature of orbital tumors are discussed.

O. SITCHEVSKA.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

ALGERNON REESE, M.D., *Chairman*

BRITTAIN PAYNE, M.D., *Secretary*

Jan. 20, 1941

The meeting was devoted to clinical evaluation of recent advances in examination and functional testing of the eyes.

Orthoptics. DR. MAYNARD C. WHEELER.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Gonioscopy. DR. JOHN M. MCLEAN (by invitation).

(The technic of gonioscopy was discussed and the normal anatomy reviewed. Various contact lenses and gonioscopes were described and the merits of different instruments and technics discussed.

Gonioscopy has usually been closely associated with the study of glaucoma, but it is also useful for study of congenital anomalies, tumors of the iris and ciliary body, cysts, foreign bodies and anterior ocular inflammations. It is valuable for studying various intraocular surgical technics.

In cases of glaucoma, gonioscopic study is helpful in planning and selecting the proper drainage operation and in studying the results of operation. It is also used to assist differential diagnosis, to measure the depth of the anterior chamber and to provide a new approach to the problem of glaucoma.

Gonioscopic photography was briefly described.)

DISCUSSION

DR. MANUEL URIBE TRONCOSO (by invitation): Dr. McLean has emphasized the importance of gonioscopy in ophthalmology and has enumerated the many diseases which occur in the angle of the anterior chamber and can be observed in the living eye. I want to stress the importance of this method for use before and after operations which open the limbus of the cornea and usually produce changes in the angle of the anterior chamber. Surgeons are beginning to realize the value of gonioscopy to show the results of their interventions and to detect many complications frequently concealed by the opaque scleral limbus.

Gonioscopy has not been accepted generally for routine examinations of the eye, owing to the necessity of using contact lenses and learning to manipulate special instruments.

The widespread use at present of contact lenses for keratoconus and for some refractive conditions has made patients more tolerant. The operator should explain to the patient that he is using a magnifying glass to observe the inside of the eye and that the patient should not squeeze the lids.

Instruments for examination of the angle must give good magnification; otherwise some lesions may be overlooked. In fact, gonioscopic examination is only a part of biomicroscopic study of the living eye. Gross changes can be ascertained with a contact glass, a binocular loupe and a flash light, but a magnification of at least 10 is necessary to obtain good images with sufficient details for an accurate diagnosis. This magnification was easily obtained with my first model of the gonioscope, but some observers found it difficult to focus the angle of the iris through the periscopic prism. The new model of the gonioscope has no periscopic prism. It was made with the cooperation of Mr. K. Larkin, and it gives a direct lateral view of the angle, has its own illuminating system and gives magnifications of 15 to 20 with exchange of eye pieces. It is a monocular instrument which gives excellent images. It is as handy and as light as the ordinary ophthalmoscope and is comparatively inexpensive. There is no objection to the binocular instrument. In fact, a stereoscopic gonioscope has been made by Mr. Larkin. He used a lighter model of the ordinary slit lamp microscope and attached to it the same coaxial illuminating system used for the monocular instruments.

The lucite contact lens is similar in outside curvature to the original glass, but the inside has a larger chamber for fluid. Plastic material makes the lens unbreakable and much lighter but it is somewhat less transparent than glass.

There are two sizes of lucite lenses, the smaller one for eyes with narrow palpebral apertures and children's eyes.

Dr. Ramon Castroviejo: When I started to work with Dr. Troncoso on gonioscopy, studying the angle of the anterior chamber comparatively in animals and in human beings, it was the aim of Dr. Troncoso to reproduce photographically the angles of different species of animals and the angle of the anterior chamber of man. The photograph taken by Thorburn is the only one known to have been taken of a human being before we started our study. The photograph shows no definition, and the details of the angle cannot be visualized. With the aid of a microscope used for metallurgic studies I was able to take photographs of the angle of the anterior chamber of a dog and a cat. The details of the angle are plainly visible. I was also able to photograph a minute piece of glass in the angle of the anterior chamber of a patient. This photograph, I believe, was the first ever taken to show with definition a pathologic process at the angle anterior chamber in a human being. However, the use of a metallurgic microscope was not practical, for obvious reasons. This instrument is expensive and is not available to the majority of ophthalmologists.

With the purpose of making simpler for the majority of ophthalmologists the visualization of the angle of the anterior chamber as well

as the photographing of it, I devised, with the help of Mr. Koch, of the Zeiss Company, an illuminating system which can be attached to a binocular corneal microscope and which may be used as standard equipment in the offices of ophthalmologists. I have two photographs in kodachrome which show a normal angle of the human anterior chamber and a melanoma at the angle of the anterior chamber. I believe these to be the first photographs in color taken of the angle of the anterior chamber in the human being. The gonioscopic equipment consists of a photographic tripod with a spring mechanism that permits an easy up and down motion. The tripod is equipped with two heads, one of which holds a binocular corneal microscope with the illuminating device. The tripod heads are assembled in such a way that the microscope can be moved in every direction, permitting a good view of the angle. When examination of the angle is performed on a patient who is lying down, the microscope can be manipulated so as to clear any protruding part of the patient. Thus, the upper half of the angle of the anterior chamber can be examined without difficulty. Once the angle is in view through the microscope, a photograph may easily be taken with any of the many 35 mm. cameras now on the market. The camera is attached to one of the tubes holding the eye piece.

Aniseikonia. DR. HERMANN M. BURIAN (by invitation).

This article will be published in full in a later issue of the ARCHIVES.

Light Threshold: Its Clinical Evaluation. DR. JACOB B. FELDMAN (by invitation).

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Angioscotometry. DR. JOHN M. EVANS (by invitation).

(The development of angioscotometry as a modern refinement in the diagnosis of obscure conditions was reviewed. Careful angioscotometric examination by the author's technic often leads to definite diagnoses and serves as an important record of progress.)

DISCUSSION

DR. MARK J. SCHOENBERG: I should like to ask Dr. Evans whether he is able to make an earlier diagnosis of glaucoma by the aid of angioscotometry than by the other known methods. Let me present a familiar example: The patient has glaucoma in one eye, and the other eye shows no evidence of pathologic change. Did Dr. Evans encounter any cases of this type in which angioscotometric examination brought out definite positive evidence confirmed by later developments? Or, take the case of a daughter or a son of a patient with chronic glaucoma. There may be vague complaints about one eye; these complaints may be forerunners of glaucoma or may be due to one of many other causes. Yet tests and examinations give negative results. Did Dr. Evans observe cases of this type in which angioscotometric examination gave conclusive results one way or the other?

Dr. Conrad Berens: In the work reported at the meeting of the American Ophthalmological Society in June, which is the study to which Dr. Evans referred, he spoke of the scotoma of menstruation. I had 1 patient who apparently did not show this. I wonder whether he has carried this work to completion and can say whether faulty technic was the cause or whether he has found other patients who do not show this phenomenon.

Dr. John M. Evans: To Dr. Schoenberg's question my answer would be "yes," except that I would not commit myself on one method of examination as a means of diagnosis of glaucoma. I believe that there is no doubt that some of my studies have shown changes in the angioscotoma long before changes actually developed in one eye, before they were detectable with the tonometer and before they were noticed in the other eye.

In answer to Dr. Berens' question, when I made my report at the meeting of the American Ophthalmological Society I thought Dr. Berens' studies were not elaborate enough to be discussed. Other studies, including observations of the blood pressure, should have been made first. The scotoma of menstruation does not exactly parallel the flow; so one would need quite a number of studies to determine whether Dr. Berens' data are complete enough to make a comparison possible.

DETROIT OPHTHALMOLOGICAL SOCIETY

Parker Heath, M.D., *President*

Edmond L. Cooper, M.D., *Recorder*

March 27, 1941

Old and New Conceptions of Stereopsis. Heinz Werner, Ph.D., Northville, Mich.

Aside from psychologic factors of experience, two general mechanisms are involved in binocular stereopsis. One is the mechanism of proprioception, i. e., the accommodation-convergence mechanism. Proprioception determines the direction and distance of objects relative to one's own body. The other is the mechanism of retinoception. Interaction between the two retinas produces stereopsis in the sense of binocular relief. The most comprehensive theory of stereopsis based on retinal interaction is that of Ewald Hering. Hering's assumptions are: 1. Corresponding points of the two retinas are geometrically defined as points equidistant from the focus. 2. Depth is directly dependent on the magnitude of retinal disparity. (Disparity is defined geometrically as deviation from strict correspondence.) I have attempted to demonstrate that Hering's "geometric" theory has to be converted into a dynamic theory in order to fit the facts. The following facts prove the dynamic character of stereopsis:

1. Binocular stereopsis is always connected with functional change of projection.

2. The disparate points of a binocularly single or diplopic image appear functionally displaced with respect to their position in the monocular field. Functional displacement toward the nasal side signifies a position in front of the fixation plane, and functional displacement toward the temporal side, a position behind it. Such displacement occurs whether the half-images are seen simultaneously or alternately in rapid succession.

3. Corresponding points are not always seen in the fixation plane. Geometrically disparate points may be perceived as lying in the frontal parallel plane. This means that under certain circumstances a change of correspondence may occur; this "secondary correspondence" signifies a change of the stereoscopic frame of reference concomitant with a "secondary disparity." "Secondary retinal correspondence" occurs frequently under normal conditions, particularly if the two images unite to a very stable binocular image. The shift of correspondence observed under abnormal conditions, particularly in cases of squint, seems closely allied to changes in retinal relations observed in experiments with normal subjects.

DISCUSSION

DR. GABRIEL STEINER: Dr. Werner has pioneered in the psychologic investigation of stereopsis. This is a problem which can be studied from three angles. The first is the philosophic angle, because three-dimensional stereopsis is related not only to the three dimensions; there is a definite time element which plays a role in stereopsis, as was clearly emphasized by Dr. Werner in his conception of interaction, or the dynamic aspect of stereopsis. Then there are the psychologic study of stereopsis and finally, the pathologic aspect. No pathologic reports of isolated stereoptic disorders associated with diseases of the brain have been mentioned to date. The lack of such reports is due not to nonexistence of such disorders but to the lack of knowledge of how to examine a patient to find stereoptic dysfunctions. Dr. Werner has laid a foundation for psychologic investigation of normal stereopsis on which ophthalmologists can rely when pathologic conditions are present.

GORDON WALLS, SC.D.: Dr. Werner's demonstration of the lability of the correspondence of retinal points is in line with certain phylogenetic considerations. Only the mammals have conjugation of the voluntary ocular movements, which is facilitated by the unique partial decussation of the chiasma. All other vertebrate classes, however, contain species with wide binocular fields, and many of these have fixative ocular movements, due to the presence of a fovea or a sharply demarcated area centralis.

I insist that all vertebrates must have single vision in their binocular fields at all times, regardless of the fact that their ocular movements are not conjugated. Therefore, the local retinal signs of direction are changed from moment to moment in accordance with the position of the eye in the orbit (or, in the case of forms with immovable eyes, such as the owl, in accordance with the position of the head relative to the body).

The ability of an occasional patient with strabismus slowly to develop a "substitute macula" having community of direction with the anatomic macula of the nondivergent eye and after operation to have a return of his ability to see codirectionally with both anatomic maculas is a vestige

of an ability which the lower vertebrates possess as a continuous and instantaneous process.

The dependence of the human being on fixed local retinal signs of direction (i.e., corresponding points) for singleness in binocular vision thus represents a phylogenetic loss of plasticity, which becomes at once evident on the development of strabismus and its accompanying diplopia. This loss of plasticity is the price which man and the mammals have paid for the motor conjugation of their ocular movements.

Treatment of Sympathetic Ophthalmia: Report of a Case. DR. JOSEPH GEMEROY.

A boy aged 6 years had a perforating injury to the right eye involving the limbus, with incarceration of the iris. Healing followed iridectomy and transplantation of a conjunctival flap. A traumatic cataract developed. One year later acute iritis developed in the injured eye as a result of a blow. The eye was sightless. Enucleation was advised, but consent was withheld by the father of the patient, although the danger of leaving the eye alone was pointed out to him. The iritis subsided in about five days. Four years later, five years after the original injury, the boy was brought in because of poor vision in the good, or left, eye. An interesting feature was the absence of inflammation in the injured eye and of pain in the sympathizing eye.

Examination of the sympathizing eye showed many cells in the anterior chamber; keratitic precipitates, and pigment deposits and exudate on the anterior surface of the lens. The iris was swollen, and the markings were indistinct. Enucleation of the injured eye was advsied because (1) it was sightless and (2) I wished to be certain of the diagnosis. Pathologic examination of sections of the removed eye proved the condition to be sympathetic ophthalmia.

The pupil was dilated at once with 10 per cent neo-synephrin hydrochloride and 0.5 per cent atropine sulfate, 1 drop three times a day from then on. This was sufficient to keep the pupil dilated except during the two periods of relapse.

Salicylates, 60 grains (3.6 Gm.) a day, were given from the beginning. This amounts to about 1 grain (0.06 Gm.) per pound of body weight. For fourteen days 4 Gm. of sulfamethylthiazole was given daily: administration of this substance was then discontinued because of a decided fall of the granulocytes in the blood. (Except for this 1 instance I have had no experience with sulfamethylthiazole.)

Under this treatment the eye showed definite improvement, the visual acuity returning from 20/40 to 20/30. Administration of salicylates and atropine was continued.

The first relapse, or flare-up occurred about four weeks after the discontinuation of sulfamethylthiazole therapy. It was characterized by formation of a mass of exudate and by precipitation of cells and pigment over the whole anterior surface of the lens. Additional treatment consisted of subcutaneous administration of diphtheria antitoxin. After making tests of the skin to determine the boy's sensitivity, I began with 1,000 units, and this was increased to 7,500 units daily for two weeks. Since with this regimen no reaction occurred, the dose was increased at the end of the fifteenth day to 15,000 units, when there developed

mild anaphylactic shock. This was what I desired. The dose was then cut to 7,500 units; it was given weekly for ten weeks and then discontinued, administration of atropine and salicylates being continued throughout this time.

The visual acuity improved to 20/30 with glasses. The precipitates disappeared from the anterior chamber as well as from the anterior surface of the lens. The dose of salicylates was then gradually decreased to 20 grains (1.2 Gm.) a day. This dosage was maintained for ten months without any untoward effects. Administration of atropine was discontinued at the end of nine months. At no time did the patient have any pain in the eye, nor was there any increase in intraocular tension.

A second flare-up occurred about three months ago (one year after onset of the original sympathetic ophthalmia). Treatment with atropine and salicylates (30 grains [1.8 Gm.] a day) was promptly begun, and this seemed sufficient to control the inflammation.

At present the boy is going to school under atropinization and is getting along well with full correction and a + 2.50 D. sphere added for reading.

I feel that the improvement was chiefly due to the use of salicylates, as recommended by Gifford, and to the use of diphtheria antitoxin, as recommended by Verhoeff.

(ED. NOTE.—Sulfamethylthiazole has not been licensed for sale in the United States and was recently withdrawn from research by the manufacturer.)

Corneal Ulcer Due to Bacillus Pyocyaneus. DR. CECIL W. LEPARD.

This paper will be published in full in a later issue of the ARCHIVES.

Book Reviews

Eames Eye Test. By Thomas H. Eames, M.D., Boston University. Yonkers, N. Y.: The World Book Co. Price, $3.50.

This test is sold as a kit consisting of (1) a visual acuity chart, (2) a +1.50 lens, (3) two stereograms, (4) a card for testing a stereoscope, (5) an astigmatic half-dial, (6) a manual of directions and (7) a sample individual record chart.

Dr. Eames has made his own compromise between a satisfactory test procedure and a simple one, and his judgment will be challenged by many. No test has yet been produced which is satisfactory as a screening test for school children and yet is simple enough to be practically applied by teachers and nurses. Possibly the Lancaster-Sloan battery of tests or set of tests provided by the American Student Health Council are subject to the least criticism.

For the sake of simplicity Dr. Eames uses only one line of test letters and one line of symbol E's. These are presumably of 6/6 or 20/20 dimensions but show unjustifiable dimensional differences. This is a fault of almost all charts that are not lithographed. He is uncritical about the illumination, specifying only "good, even illumination (*not* direct sunlight)." He will find many supporters for his choice of a one line test chart, but there will be an equal number of dissenters. Since no visual acuity test is adequate to uncover moderate but significant degrees of hyperopia or astigmatism, he includes a +1.50 sphere and an astigmatic dial to help in weeding out these defects. The use of the sphere is justified but has usually been met with disapproval either by those who object to it as doubling the work involved or by those who claim that it is inadequate. Likewise, the use of a stereoscope as a test for fusion and coordination has met with so much resistance that the Eye Health Committee rejected it.

Dr. Eames has written an admirably simple manual to go with his test kit and to explain the performance and scoring of the test. It includes, among other things, a list of twenty-four common ocular symptoms. For an alert observer this list is probably as valuable as the test procedures (or almost any other "practical" battery of tests) in discovering handicapped school children.

LE GRAND H. HARDY.

Tuberculinothérapie dans les névralgies faciales idiopathiques et certaines affections essentielles (Investigation clinique). By Dr. Carlos Charlin C., Professeur á la Faculté de Médecine. Pp. 336. Nascimento, Santiago, Chile, 1940.

The first observation is a reprint of a case of corneal ulceration with persistent severe pain and rhinorrhea reported in the *Annales d'oculistique* in 1931. The manifestations led to the recognition of a lesion of the nasal nerve, a branch of the ophthalmic nerve, and a syndrome was established which bears the name of the author. Examination of the nose reveals marked congestion and swelling of the anterior tip

of the turbinals, and the eye shows a lesion of the anterior pole: corneal ulcer, iritis or keratitis. The pain which is characteristically severe and the other symptoms are relieved by nasal treatment with cocaine and epinephrine. Another symptom is tenderness of the skin of the nose near the nostril and the inner canthus. All of the symptoms of the syndrome may not be present. The author then goes into the question of causes; in addition to a local cause in the nose he mentions other causes and reports 2 cases of glandular tuberculous toxemia in which after a diagnostic Mantoux test with a 1 : 10,000 solution of tuberculin the neuralgic pain disappeared. The patients also received inunctions of ectebine and injections of methylic antigen of Nègre and Boquet.

The results in these 2 cases led the author to investigate other forms of neuralgia, which he treated with success, and he concludes that in cases of idiopathic facial neuralgia and of trigeminal neuralgia in which the cause is unknown tuberculin treatment should be tried. In conclusion he states: "One must remember that tuberculous infection is a normal infection in the human race and that it can produce quite ordinary lesions without any specific characteristics."

ARNOLD KNAPP.

Conos Y Bastoncitos. By Jose Sverdlick (Doctor's thesis). Pp. 109, with 82 illustrations. Buenos Aires, Argentina: Aniceto Lopez, 1940.

The first part of the monograph by Sverdlick on cones and rods consists mostly of a comprehensive review of the comparative anatomy of these structures in invertebrates and vertebrates. In addition, it contains the results of original microscopic examinations by the author of the neuroepithelium of primates, ungulates, rodents, aves and reptiles. The ontogenetic development of rods and cones in human embryos of 25 to 200 mm. crown-rump length is treated in the second part of the monograph. The third and last part considers the fully developed human retina.

The author used a great variety of stains, including several silver impregnation methods. There are eighty-two illustrations, the majority of which are photomicrographs. Most of them are instructive; others fail to fulfil their purpose.

The observations of the author confirm previous reports of the cones and rods of many species. Of greater value, however, are several new descriptions of animals native to South America.

L. v. SALLMANN, M.D.

Directory of Ophthalmologic Societies *

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President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
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All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

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Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. A. J. Ballantyne, 11 Sandyford Pl., Glasgow, C. 3, Scotland.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive.
All correspondence should be addressed to the President.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 Ill tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: Atlantic City.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank R. Spencer, Physician's Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-23, 1941.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Allen Greenwood, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. John Evans, 23 Schermerhorn St., Brooklyn.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Vaillancourt, 46 St. Louis St., Quebec.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. V. May, 1703 Main St., Marinette.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Marinette. Time: November 1941.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Spencer S. Howe, 103 E. Holly St., Bellingham, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 419 Stimson Bldg., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. E. Rideout, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. Kessler, 311 Center Ave., Bay City, Mich.
Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Davis, 1615 Howard St., Omaha.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Franklin P. Maury, Professional Bldg., Tucson, Ariz.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. J. K. Heckert, 103 N. Washington St., Lansing.
Secretary-Treasurer: Dr. W. D. Irwin, 710 Hanselman Bldg., Kalamazoo.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. H. Bailey, 29 S. Oakland Ave., Sharon.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. W. E. McClellan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.
Place: Macon. Time: May 13-17, 1941.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. K. Von Lackum, 117–3d St. S.E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGearv, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. A. L. Hammerel, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.
Place: Atlantic City. Time: June 3-5, 1941.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Secretary: Dr. Vanderbilt F. Couch, 105 W. 4th St., Winston-Salem.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. E. Spear, Dickinson.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Roderick Macdonald, Rock Hill.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia Hotel, Columbia. Time: November 1941.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. E. L. Goar, 1300 Walker Ave., Houston.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.
Place: San Antonio. Time: December 1941.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 628 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. George G. Hawkins, Newport News.
Secretary-Treasurer: Dr. Guy Fisher, 3 E. Beverley St., Staunton.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Black, 7 W. Bowery St., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Sanford Gifford, 720 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Wallace W. Ralston, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. John I. Garret, 57 Stokes Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Paul Southgate, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kirt G. Parks, 605 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Harold Mulligan, 1680 N. Vine St., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Leonard C. Whiting, 121 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Algernon Reese, 73 E. 71st St., New York.
Secretary: Dr. Brittain Payne, 896 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. James W. Smith, 1016–5th Ave., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Harold Gifford, 1620 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Secretary: Dr. Wilfred E. Fry, 1819 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward A. Weisser, 119–5th Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. J. Avery, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 5 p. m., Jan. 22. 1941.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William M. James, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. H. Rommel Hildreth, 823 Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fred Boyle, 490 Post St., San Francisco.
Secretary: Dr. Frank Hand, 450 Sutter St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. R. Webster, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. John R. Myers, State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. S. H. Patterson, 1251–15th St., Toledo, Ohio.
Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Sterling Bockoven, 1752 Massachusetts Ave. N.W., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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